THE Journal of the Kansas Medical Society

OCTOBER
1956

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NO X

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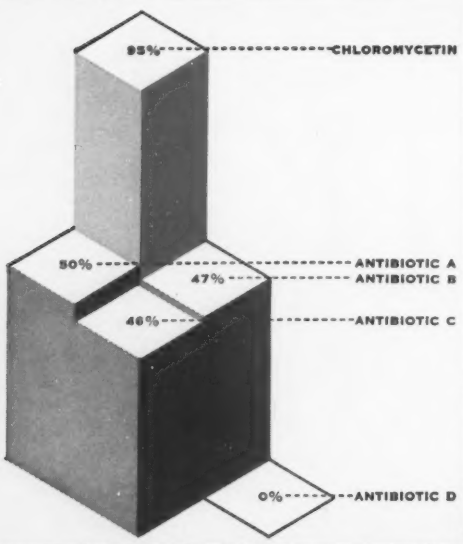
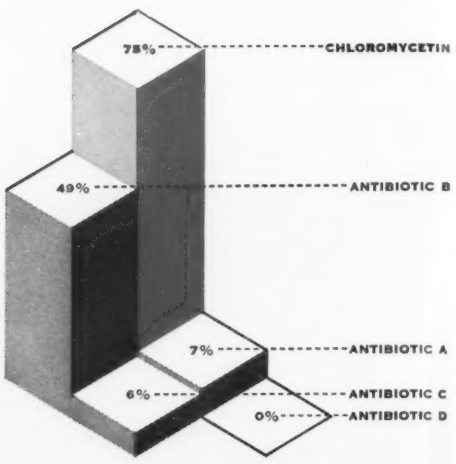
SENSITIVITY OF COMMON PATHOGENS TO CHLOROMYCETIN AND FOUR OTHER MAJOR ANTIBIOTIC AGENTS*

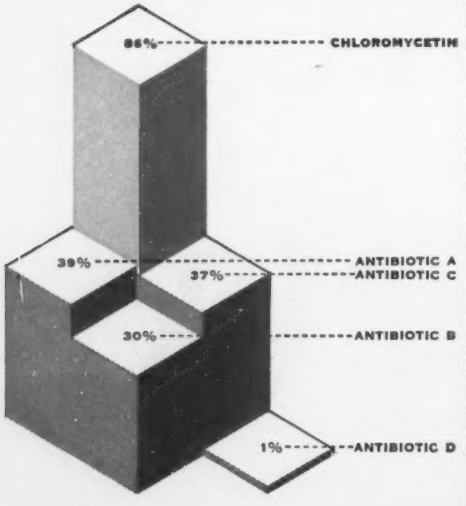


ESCHERICHIA COLI
(148-227 STRAINS)

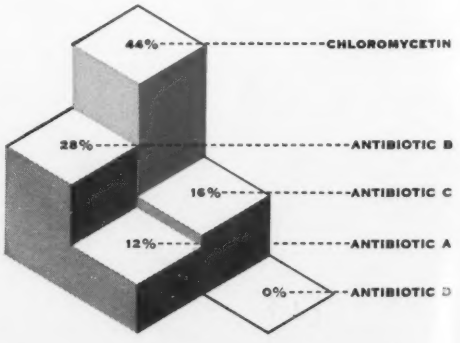


BACILLUS PROTEUS
(63-104 STRAINS)

AEROBACTER AEROGENES
(143-248 STRAINS)



PSEUDOMONAS AERUGINOSA
(39-70 STRAINS)



*This graph, based on *in vitro* studies, is adapted from Horton and Knight.[4]

when more than one organism is involved...

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for today's problem pathogens

Therapeutic advantages of CHLOROMYCETIN (chloramphenicol, Parke-Davis) are especially appreciated when mixed infections are encountered because it provides highly effective antibiotic action both against gram-negative and against gram-positive pathogens.[1-7] CHLOROMYCETIN also acts against many pathogens which may grow when originally sensitive organisms have been suppressed.[2]

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References: (1) Felix, N. S.: *Pediat. Clin. North America* 3:317, 1956. (2) Joron, G. E.; Fowler, A. F.; de Vries, J.; Reid, G., & Mathews, W. H.: *Canad. M. A. J.* 73:956, 1955. (3) Weil, A. J., & Stempel, B.: *Antibiotic Med.* 1:319, 1955. (4) Perry, R. E., Jr.: *North Carolina M. J.* 16:567, 1955. (5) Jones, C. P.; Carter, B.; Thomas, W. L., & Creadick, R. N.: *Obst. & Gynec.* 5:365, 1955. (6) Murphy, F. D., & Waisbren, B. A., in Murphy, F. D.: Medical Emergencies: Diagnosis and Treatment, ed. 5, Philadelphia, F. A. Davis Company, 1955, p. 557. (7) Altemeier, W. A.; Culbertson, W. R.; Sherman, R.; Cole, W.; Elstun, W., & Fultz, C. T.: *J.A.M.A.* 157:305, 1955. (8) Horton, B. F., & Knight, V.: *J. Tennessee M. A.* 48:367, 1955.

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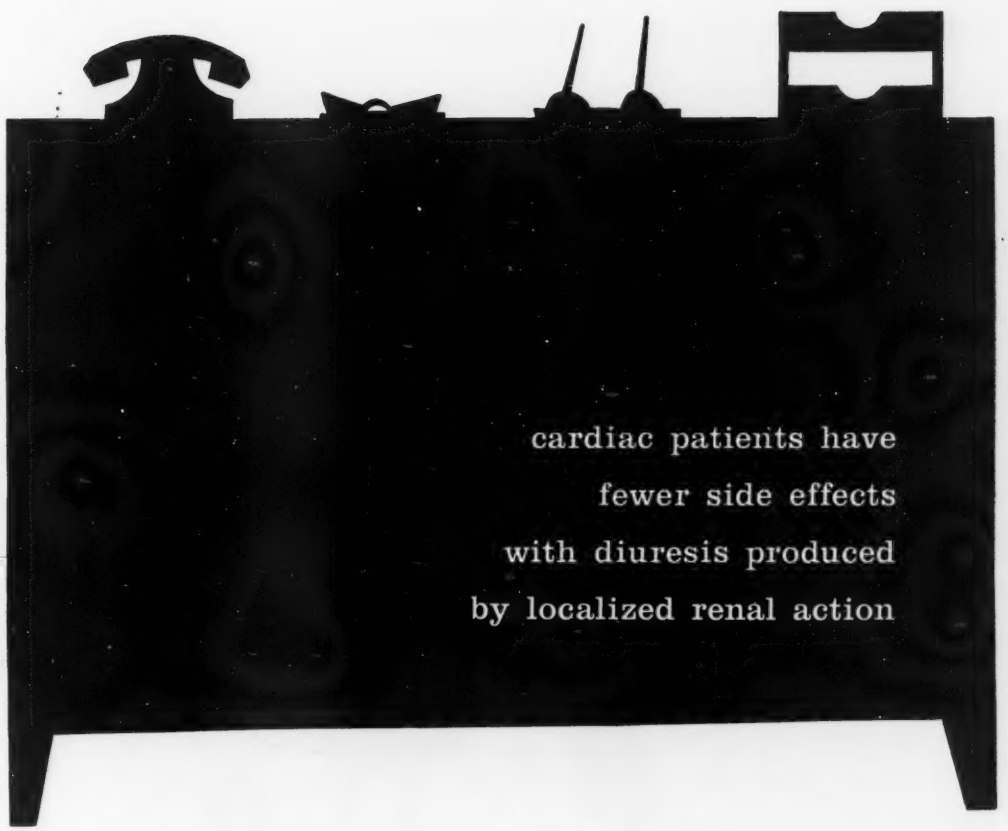

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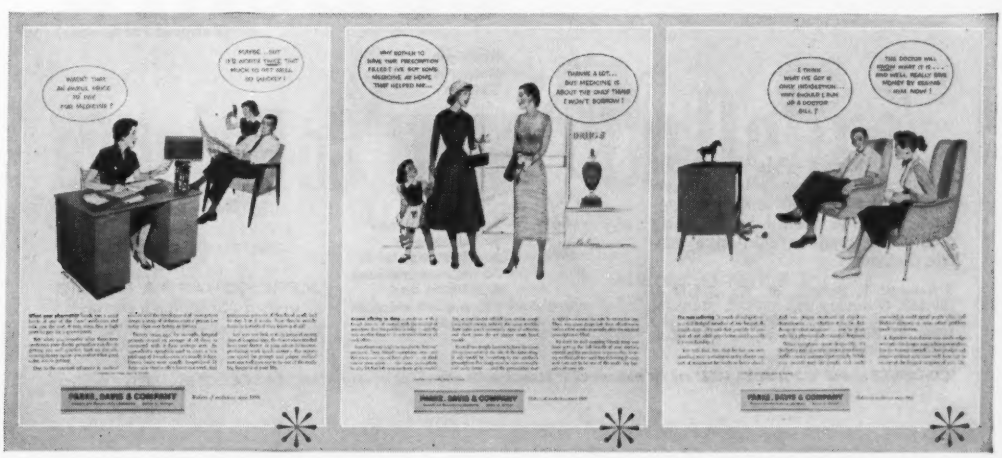

in inflammatory skin diseases
all the benefits of the "predni-steroids"
plus positive antacid action
to minimize gastric distress

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'Co-Deltra'
(Buffered Prednisone)

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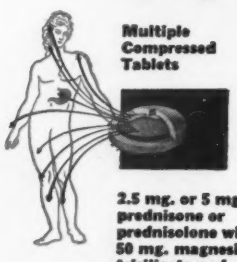

'Co-Hydeltra'
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2.5 mg. or 5 mg. prednisone or prednisolone with 50 mg. magnesium trisilicate and 300 mg. aluminum hydroxide gel.

Clinical evidence[1,2,3] indicates that to augment the therapeutic advantages of prednisone and prednisolone, antacids should be *routinely* co-administered to minimize gastric distress.

References: 1. Boland, E. W., *J.A.M.A.* 160:613, (February 25,) 1956. 2. Margolis, H. M. *et al*, *J.A.M.A.* 158:454, (June 11,) 1955. 3. Bollet, A. J. *et al*, *J.A.M.A.* 158:459, (June 11,) 1955.

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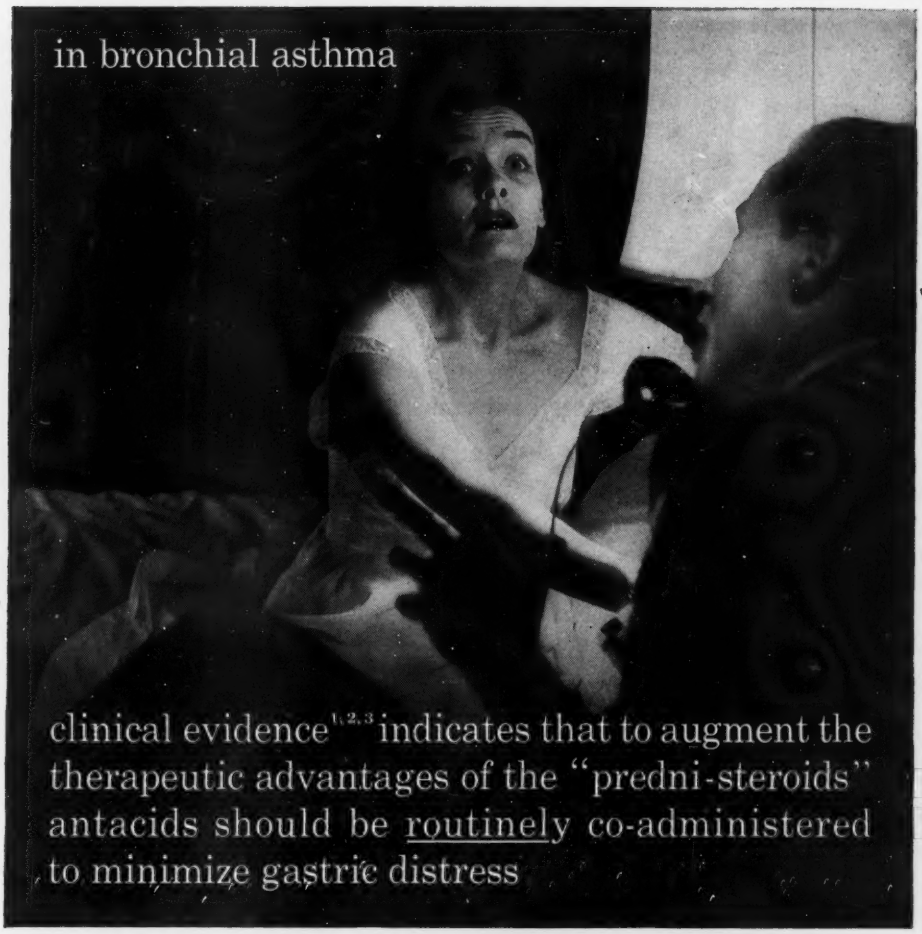
in bronchial asthma
clinical evidence[1,2,3] indicates that to augment the therapeutic advantages of the "predni-steroids" antacids should be routinely co-administered to minimize gastric distress

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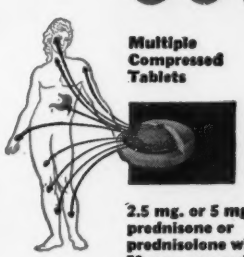
'Co-Hydeltra'
(Buffered Prednisolone)



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(Buffered Prednisone)

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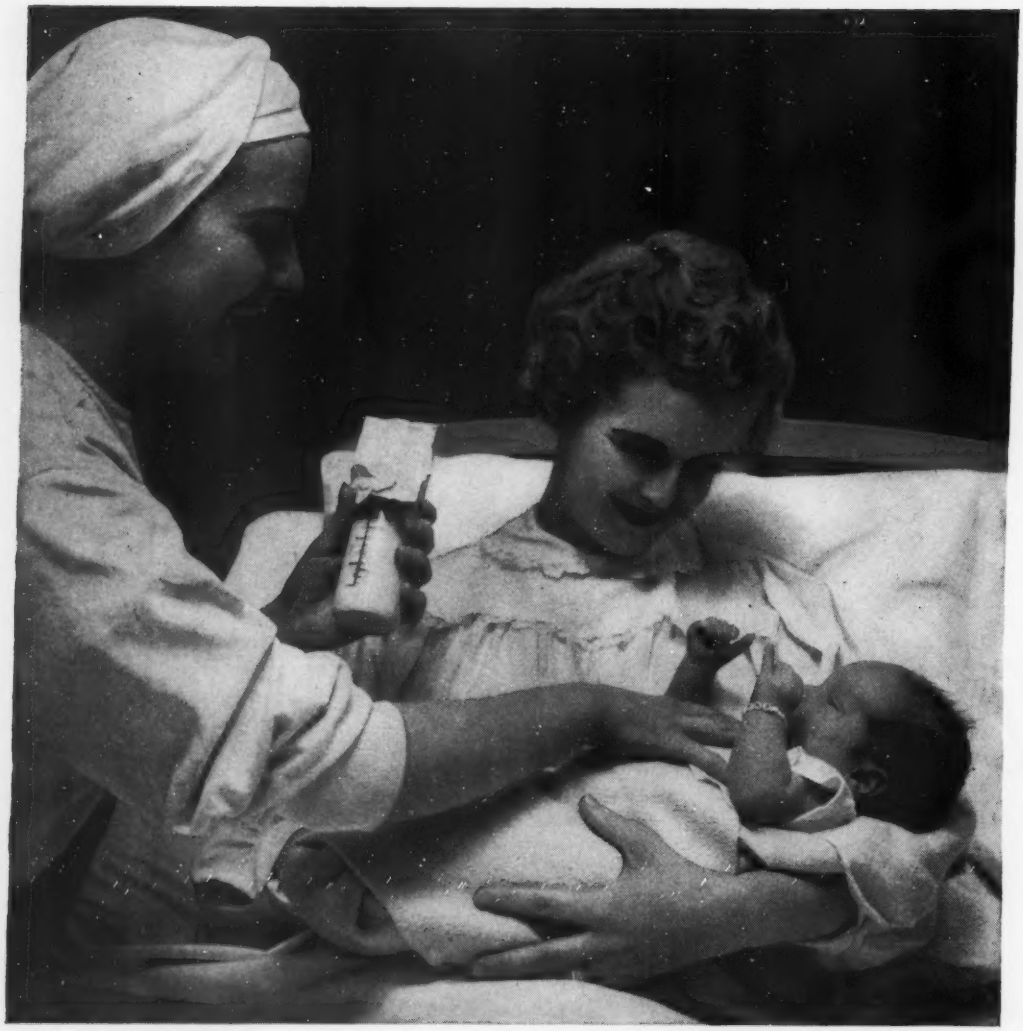

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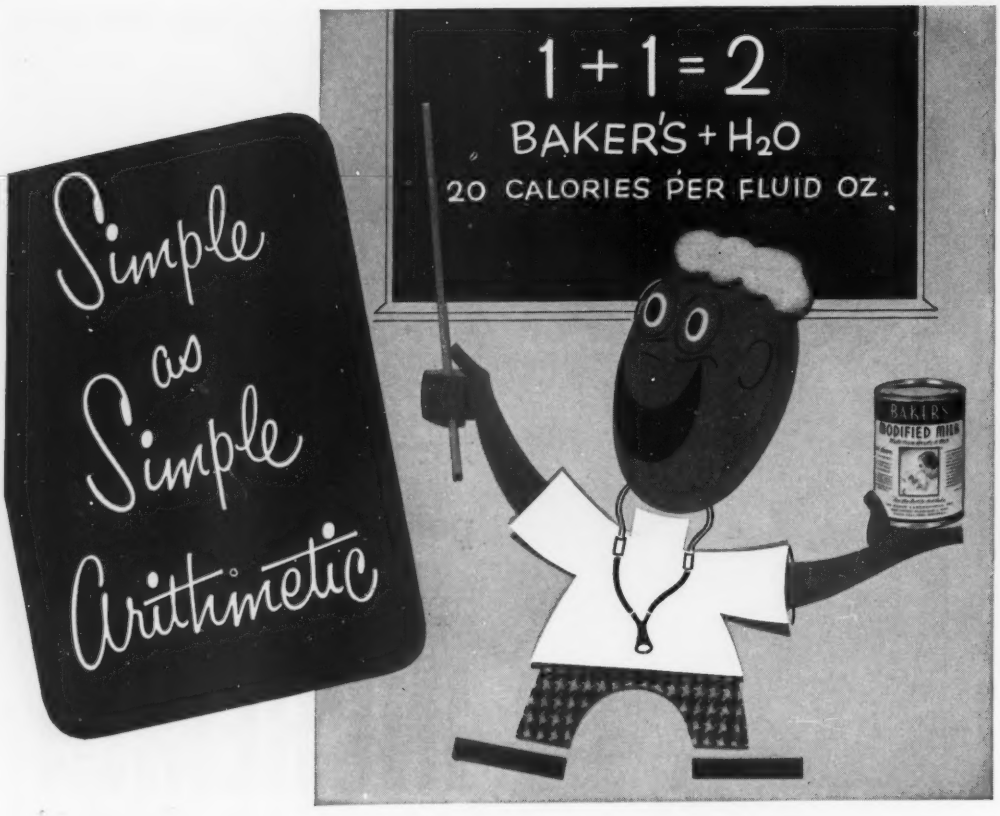

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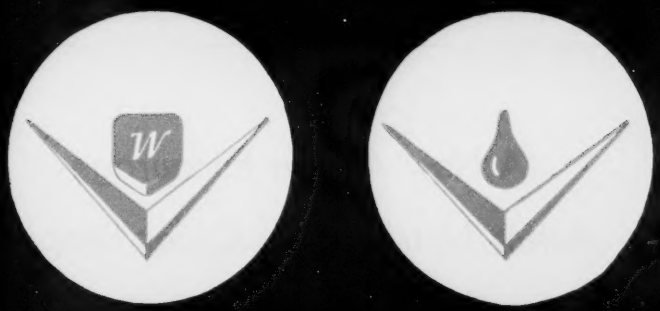

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1. Martin, W. J., et al.: J.A.M.A. *160*:928 (March 17) 1956.

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Vitamin B_2	.79 mgs.
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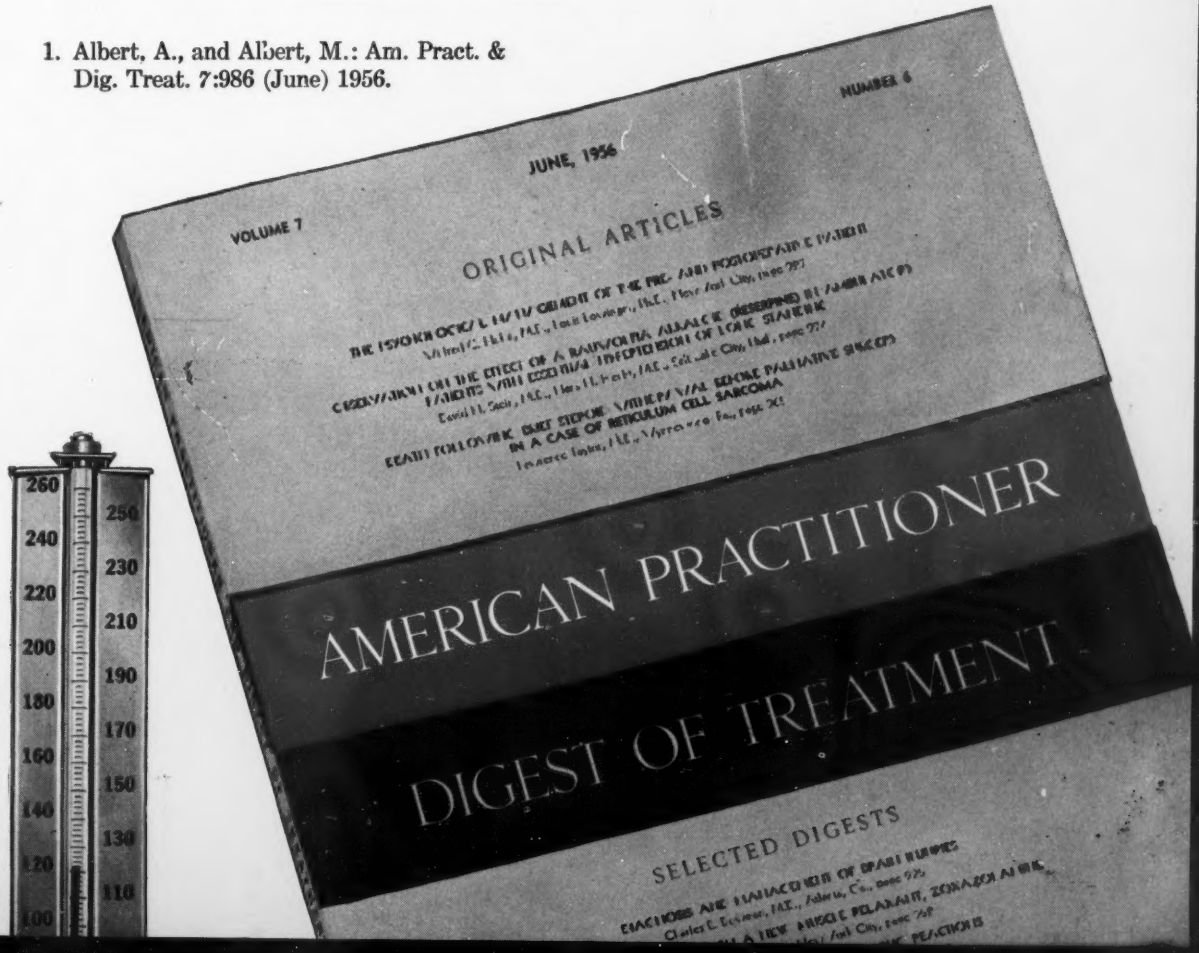
1. Albert, A., and Albert, M.: Am. Pract. & Dig. Treat. 7:986 (June) 1956.



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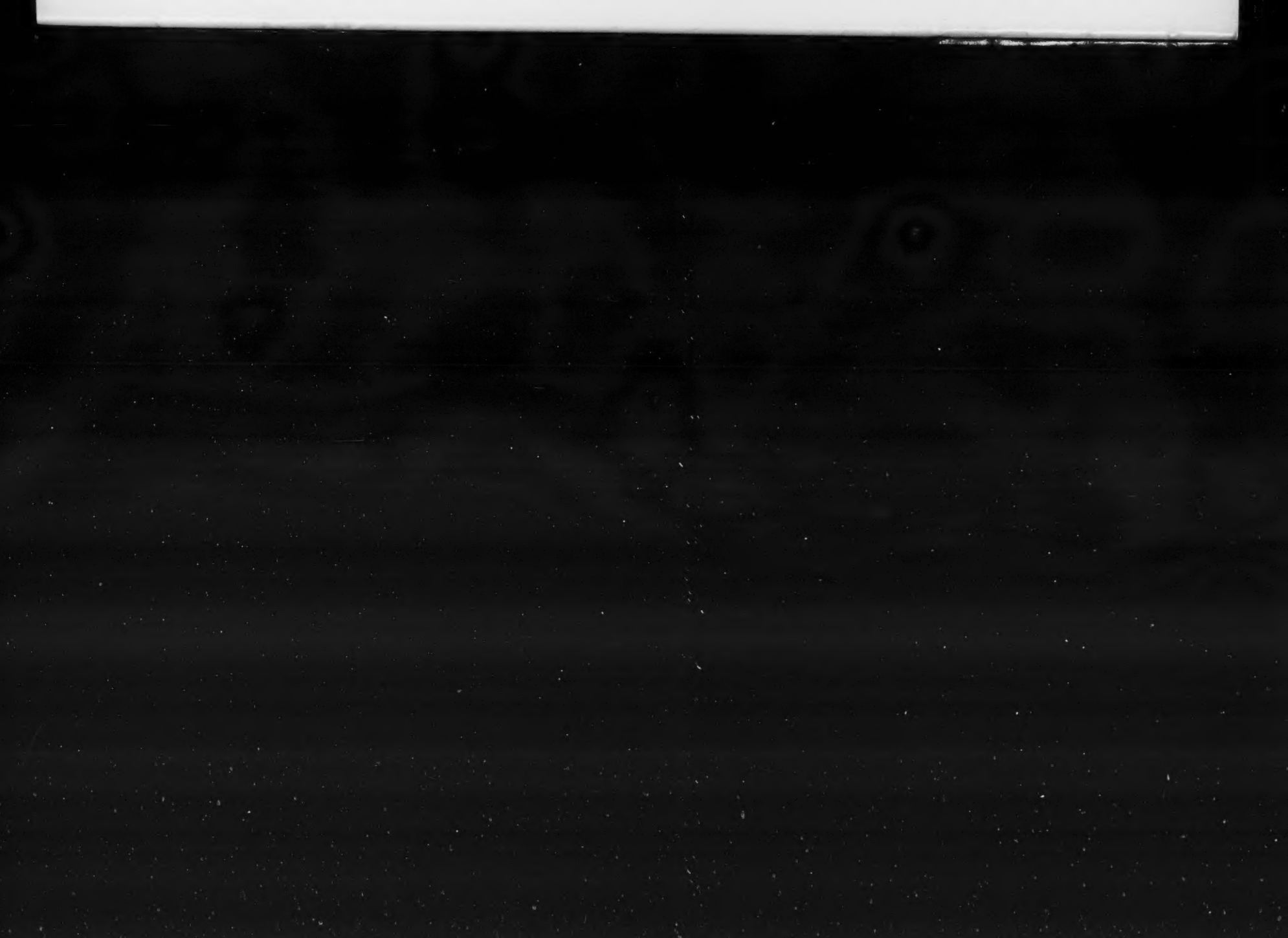

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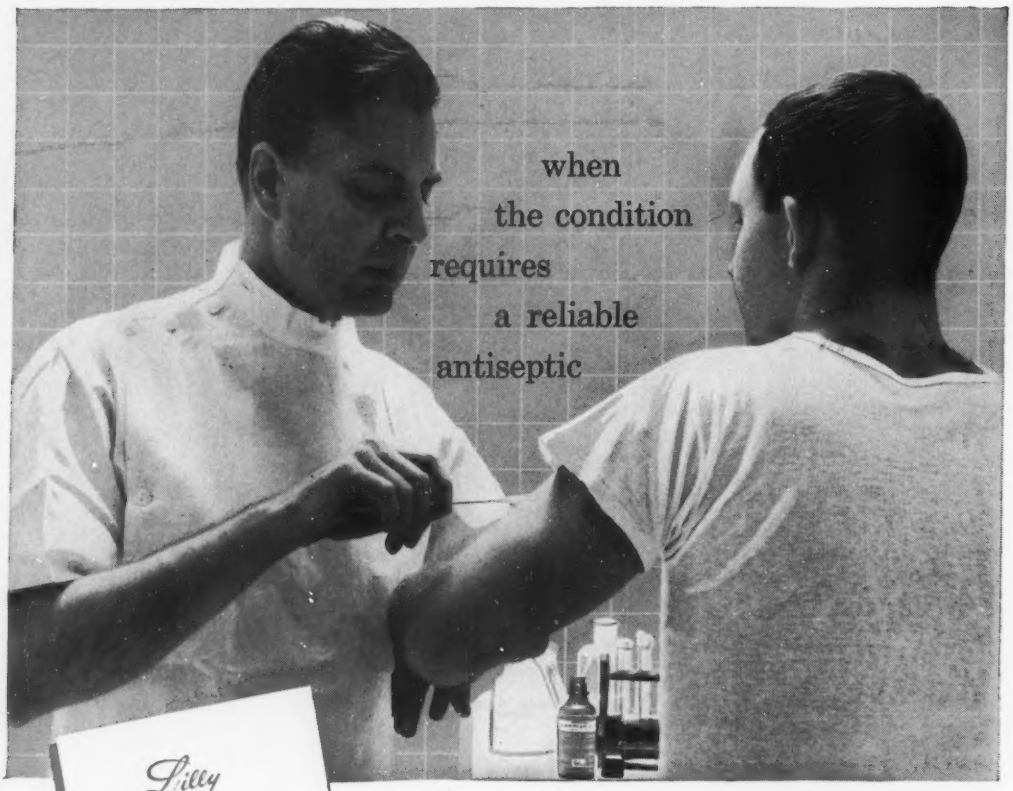

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Gamma Globulin Deficiency

Report of a Case and Survey of the Literature

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Agammaglobulinemia is a recently described disease entity consisting of unusual susceptibility to frequently recurring infectious episodes associated with low or absent levels of gamma globulin in the blood. Typical clinical and laboratory findings are presented, and the treatment of one case is discussed.

A new disease of unusual interest, manifested by a marked decrease or absolute deficiency of gamma globulin in the blood and a heightened susceptibility to infection, has been described with increasing frequency in recent years. This syndrome, variously named hypogammaglobulinemia, agammaglobulinemia, or globulin deficiency disease, is of utmost importance because of its basic and far-reaching implications to the field of immunology and to the pathologic physiology of the body's responses to infectious processes.

To the research worker a study of this entity affords an unusual opportunity to delve further into many of the unsolved problems of the body's immune mechanisms. To the clinician it is an excellent example of the pathophysiologic approach since its many laboratory and clinical features can easily be deduced from a knowledge of the basic defect. Judging from the many reports and discussions in the literature since the original case was recorded only four years ago by Bruton,[1] this certainly is not a rare condition, and its recognition is important since it doubtlessly explains many puzzling and obscure clinical pictures which are not otherwise well understood.

The patient described in the following report was observed recently at the University of Kansas Medical Center. His history is of interest since it is typical of the usual findings and serves to illustrate the major features of this condition.

CASE REPORT

A 58-year-old white male was first admitted to the University of Kansas Medical Center in March of 1953 complaining of fever and cough and stating that he had had innumerable episodes of "pneumonia" for about 35 years. He said that his most recent difficulties had been in December of 1952, February of 1953, and just prior to this hospitalization. During each one of these episodes he had had fever, malaise, weakness, and a productive cough. He received intermittent therapy with penicillin for several years, usually with good results. He claimed that he had shown no unusual susceptibility to infections prior to 35 years before, at which time he developed bilateral ear infections. Since then he had had repeated episodes, as described, but had felt well between febrile periods and had worked at various clerking jobs. No members of his family had shown similar tendencies.

The main physical findings at admission were a temperature of 104.4 degrees, an appearance of being both acutely and chronically ill, and moist

rales over the right lung posteriorly. The liver was palpable 4 cm. below the right costal margin and was soft and non-tender; the spleen was felt 2½ cm. below the left costal margin. There were no other important physical abnormalities.

Laboratory work revealed a 1-plus albuminuria but an otherwise normal urinalysis. Red blood count was 3,300,000, hemoglobin 10.3 grams, white blood count 2300 with 74 polys (8 non-filamented), 24 lymphocytes and 2 monocytes. Serology was negative; non-protein nitrogen was 33 mg. per cent; blood sugar was 90 mg. per cent. Skin tests were negative for tuberculosis, histoplasmosis, blastomycosis, and coccidioidomycosis. A congo red test showed 80 per cent of the dye remaining in the blood; phenol red excretion and urea clearance were normal. Agglutinations for typhoid, paratyphoid, brucella, and tularemia were negative in all dilutions, as were cold agglutinins. Repeated blood cultures were negative, and nose and throat cultures showed the "usual flora."

Urine cultures showed a moderate growth of paracolon bacillus, and sputum cultures showed E. coli and hemolytic staphylococcus. Sedimentation rate was 32 mm. in one hour. Liver function tests showed a total serum bilirubin of 0.7 mg. per cent, alkaline phosphatase of 1.6 millimol units, prothrombin time 65 per cent of normal, bromosulphalein retention of "0," cephalin cholesterol flocculation 4-plus, thymol turbidity of 25 units, serum albumin of 4.2 gm. per cent, and serum iron of 54 gamma per cent. Bone marrow showed a neutrophilic hyperplasia of marrow tissue with a moderate increase in monocytes and monocytic-like cells. A liver biopsy revealed only mild acute and chronic hepatic changes. A lymph node biopsy was reported as showing lipomatosis. Bronchoscopy and bronchial washings revealed no malignant cells.

An electrocardiogram was normal, and a chest x-ray revealed an increase of the hilar shadow bilaterally, being more prominent on the right, and infiltrative changes in the base of the right upper lobe with some elevation of the right interlobar septum. Upper and lower gastrointestinal series were normal. A bronchogram revealed no major abnormalities, and an intravenous pyelogram revealed only minor changes consistent with minimal right pyelonephritis.

The patient's hospital course was one of continued fever controlled initially with aspirin. On the fifth hospital day he developed lower left chest pain, and at that time a chest film showed lower left lobe pneumonia. Penicillin and streptomycin were started, but the fever continued through the 23rd hospital day and there was no change in his general condition. At that time cortisone and Chloromycetin were begun, and his fever subsided shortly thereafter. These medications were gradually withdrawn and were discontinued on the 32nd hospital day. Clinical improvement continued, and he was discharged on the 37th hospital day without any specific diagnosis having been made.

The patient's second University of Kansas Medical Center hospitalization occurred in March of 1955, almost exactly two years after his initial work-up. On this admission he stated that he had been in reasonably good health since his previous hospitalization except for an occasional short febrile bout. About two weeks prior to admission he started running a fever and was treated with Aureomycin, 250 mg. four times daily, and within a few days became afebrile until the day prior to his hospitalization. Physical examination revealed a well developed, well nourished white male who appeared chronically ill. His temperature was 102 degrees, and he had a marked seborrheic dermatitis involving his scalp and face. The liver was palpable 3 cm. below the right costal margin, and the spleen was felt 2½ cm. below the left costal margin. The lung fields were clear, and the heart was normal to examination. There were no palpable lymph nodes and physical examination was otherwise unremarkable.

His red blood count was 3,500,000, white blood count was 7300 with a differential count of 69 polys (17 non-filamented), 21 lymphocytes, and 10 monocytes. Blood chemistries were within normal limits, serology was negative, and the glucose tolerance curve was normal. Agglutinins for typhoid, paratyphoid and brucella were negative in all dilutions.

Urine cultures were negative, and repeated sputum cultures failed to grow out any significant organisms. Heterophile and cold agglutinins were absent, and blood cultures were negative on several occasions. Sedimentation rate was 14 mm. in one hour. An electrocardiogram was within normal limits. Bone marrow biopsy was reported as showing an increase in monocytes and monocytic-like elements and a shift to the left in neutrophilic elements. Liver function profile revealed a serum bilirubin of 1 mg. per cent, total cholesterol of 240 mg. per cent with 54 per cent esters, serum albumin of 4.9 gm. per cent, serum globulin of 0.46 gm. per cent and bromosulphalein retention of 21 per cent. Thymol turbidity and cephalin cholesterol flocculation were negative.

Electrophoretic studies of his blood failed to show the presence of gamma globulin. A careful study of his blood hematologically failed to show isohemagglutinins. Typhoid immunization was attempted, and he responded to each injection with marked temperature spike, but repeated studies in ensuing weeks failed to reveal any agglutinins to typhoid. The patient was febrile during the early part of his hospital stay until he was given an injection of 20 cc. of gamma globulin, at which time his temperature

subsided almost dramatically. He remained afebrile except for the temperature elevations with typhoid immunization and felt well on discharge from the hospital. Since then, he has been followed in the Kansas University Medical Center Out-Patient Clinic and has had a few febrile bouts, each of which responded promptly to gamma globulin injections.

HISTORY AND CASE REPORTS

The first recorded case in which a typical clinical syndrome was correlated with a decrease of gamma globulin in the blood was reported by Bruton[1] in 1952 in an eight-year-old boy who had had repeated blood stream and respiratory infections for four years. On ten separate occasions a pneumococcus had been recovered from his blood. This child had mumps at three separate times and failed to develop a negative Schick test after repeated injections of diphtheria toxoid. He was also found to be unable to produce antibodies against typhoid vaccine and pneumococcal polysaccharide. Electrophoretic studies showed a complete absence of gamma globulin in his blood, and intermittent injections of gamma globulin resulted in both symptomatic and clinical improvement.

Numerous other reports have followed in the last four years with a steady stream of descriptions of this disease. It is now apparent that this is probably not a rare condition but one awaiting only greater awareness by the clinician to make its recognition relatively common. Of the more recent reports, Janeway has described nine boys with agammaglobulinemia and suggested that this was sex linked and a congenital defect.[2]

An early report of this entity involving a female was that of an eight-weeks-old girl who did not develop neutralizing antibodies to vaccinia and who died of progressive vaccinia. Her blood showed no gamma globulin, and autopsy showed a striking absence of lymphocytes in lymph nodes and spleen, correlating with clinical lymphopenia.

This entity has been described in a 58-year-old female with malignant lymphoma, indicating its presence in other disease states. Various authors have stressed the frequency of bronchiectasis in adult cases. Splenomegaly has been frequently cited in these patients, with an accompanying leukopenia. Various other interesting case histories have also been recorded, and all have stressed the virtual lack of resistance to infection with an accompanying grossly deficient blood level of gamma globulin. Most of these reports deal with the fascinating clinical ramifications of this problem and include speculation as to the yet unknown origin of the underlying disease problem.

PHYSIOLOGY OF THE IMMUNE MECHANISM AS RELATED TO GAMMA GLOBULIN

One of the reasons for the tremendous amount of interest in this disease is that it certainly gives proof to the long suspected belief that gamma globulin is closely related to the mechanisms of resistance to infection. However, unfortunately, there are many gaps in our knowledge about gamma globulin itself.

It has been calculated that approximately 13 per cent of the total serum proteins are gamma globulin, which is chiefly concerned in the immunological process, but a small portion of the gamma globulin fraction is, however, of unknown significance. Careful studies have demonstrated that there are various subfractions of the gamma globulin with each portion having antigenic activity, that their precipitability with different antigens varies, and that there presumably are inhibiting fractions which can block precipitation of one sub-group while enhancing precipitability of another. Antibody titers are said to be the result of competing protein molecules of varying reaction capacity. However, not all antibodies are in the gamma globulin group, for blood group isohemagglutinins, typhoid "O" agglutinins, and true Wassermann reagin are said to be beta-2 globulins.

The normal adult has 25 mg. of circulating gamma globulin in dynamic equilibrium with a similar amount which is located extravascularly, and the serum levels are closely related to adaptive processes. They are known to be high at birth because of the transplacental transmission of antibodies, fall for several months, and thereafter rise slowly as the individual comes in contact with immunizing diseases. This low gamma globulin in infancy seems well correlated with the fact that the early months are the precarious ones as far as infectious diseases are concerned, and many clinicians feel that treatment with pooled gamma globulin protects against infectious episodes in young children as well as in others with refractory infections. Studies with the administration of radioactive gamma globulin (tagged with I^{131}) have shown that half life is approximately 13 days in adults and 20 days in children and that the normal adult apparently catabolizes one-third of his total supply (about 3.8 grams) daily.

Perhaps the greatest defect in our knowledge of the globulins is related to their site of origin. It would seem that the existence of this disease with its immunoglobulin defect would point to a special site and mechanism for its synthesis. Various workers favor the reticulo-endothelial cells, immature tissue lymphocytes, plasma cells, or a combination of these. There is favorable evidence for the plasma cell as a site of gamma globulin production since it has been shown that in hyperimmune rabbits there is a massive

plasma cell infiltration of many organs, and the high gamma globulin levels which accompany this state fall with cortisone-induced regression of these cells. In children with agammaglobulinemia, regional lymph nodes show no germinal center formation or plasma cells after antigenic stimulation, whereas this is not so in normal children. In multiple myeloma, a disease involving plasma cells, aberrations of gamma globulin have been reported. Careful bone marrow studies in patients with agammaglobulinemia seem to point toward a plasma cell deficiency as compared to normal persons, and the same is true in reported investigations of lymph node histology. In persons with rheumatic fever, in which elevated gamma globulin levels frequently are encountered, plasmacytosis is often seen.

However, there is also some fairly impressive evidence implicating fixed tissue lymphocytes which, it is suggested, may become released as blood lymphocytes or while still in lymphoid tissue may undergo cytolysis and give rise to blood antibodies by shedding their surface protein films. To support this view, several authors cite lymphopenia in patients with agammaglobulinemia. They also point out that histologically lymph nodes tend to be hypoplastic, which raises a strong temptation to correlate the absent gamma globulin, blood lymphopenia, and quantitative lack of tissue lymphocytes at biopsy and to interpret them as being due to greatly reduced lymphopoietic activity. In refutation of this idea, however, is the knowledge that not all persons with this disorder show definite evidence of reduced lymphocytic activity.

Of some importance is the fact that factors other than gamma globulin are related to the body's ability to resist infectious episodes, and these must be considered in the evaluation of individuals who seemingly have an inability to cope with infection. It has been pointed out that local anatomical changes, including diminished blood supply and obstruction to hollow viscera, changes in tissue reactivity as might occur in various metabolic disorders (scurvy), and failure of normal defense mechanisms as might occur under heavy anesthesia or with neutropenia, all vitally influence the host's response to a bacterial or viral invasion. Of great interest along the same lines is a newly discovered serum protein, properdin, which seems to act with complement and magnesium and seems to participate in such reactions as destruction of bacteria, neutralization of viruses, and lysis of certain red blood cells.

ETIOLOGY OF GAMMA GLOBULIN DEFICIENCY

There has been no really adequate explanation of the etiology of this disorder, a fact which probably is to some extent related to our deficiency in knowledge of the basic physiology of gamma globulin. It is important to appreciate that low levels of gamma globulin have been recognized in various disease states, such as severe nutritional deficiency, the nephrotic syndrome, multiple myeloma, in states associated with marked protein catabolism, in so-called idiopathic hypoproteinemia, and in the face of inadequate lymphoid tissue function, such as might occur with cancerous replacement and radiation damage to lymphatic tissues. These conditions differ from the disease described in this paper since they are associated with low total proteins of which gamma globulin deficiency is only a part.

From a clinical point of view it would seem likely that there are numerous causes of gamma globulin, many of which are not at all understood as yet. Most authors are inclined to divide their cases into acquired and hereditary. The latter type was brought to mind almost immediately since early case descriptions were all noted in male children. Many authors soon believed that agammaglobulinemia was a sex-linked recessive condition, analogous in its mode of transmission to hemophilia, with the basic defect being an inability to synthesize gamma globulin. However, it was not long before an increasing number of case reports began to appear describing this disease in adults of both sexes who had clearly had their afflictions for only a few years. At this point it became feasible to describe a secondary or acquired form of this entity which seemed to develop at any age and in either sex.

It is felt that in some of the acquired cases the globulin deficiency is secondary to a definite disease process which involves the lymphatic or reticuloendothelial system, such as leukemia, sarcoidosis, Hodgkin's disease, histoplasmosis, et cetera. Most of the so-called "acquired" cases which have been described, including the one outlined in this paper, are not associated with any discernible underlying disease state. This makes appreciation of their etiology a rather difficult matter.

An interesting hypothesis has been presented by Bram and Morton[29] who find it difficult to envision any acquired process that would selectively assault globulin production without affecting closely related functions. They feel that elimination of a single protein is much more characteristic of a hereditary process in which the gene for that particular function is defective. They also point out that the late appearance of this process in some patients is not inconsistent with their theory, particularly when it is considered that many inherited defects make their appearance at varying ages.

It too is their belief that some of these "secondary" cases described in lymphomas probably were not associated with a lymphoma in so far as pathologists

at times have great difficulty in distinguishing a primary neoplastic change from the intense reticuloendothelial hyperplasia that can occur in response to agammaglobulinemia.

Furthermore, they believe that cases described as being due to a granuloma or other infectious processes showed that more than likely the infection that was present was related to the patient's poor resistance due to globulin deficiency because it seemed unlikely that a granulomatous process could involve globulin producing centers alone. Their reasoning sounds logical, and it would seem that the hypothesis they present has much merit. However, in most studies little clinical evidence can be obtained showing a familial incidence, and examinations of relatives of patients with gamma globulin deficiency have revealed no globulin defects.

It has been suggested that perhaps gamma globulin deficiency could be related to some defect in the pituitary adrenal axis. Good and Kelly[37] studied seven patients with this disorder to see if they could ascertain any adrenal-cortical dysfunction, but with careful laboratory examination they could find nothing to support such a concept. Martin[34] raised another interesting point relative to the etiology of this disorder by studying the blood proteins of four premature infants, and he discovered an almost total lack of gamma globulin at or before the 26th week. It is his belief that agammaglobulinemia represents in some manner a persistence of the fetal pattern.

NATURE OF THE GLOBULIN DEFECT

In the absence of any definite knowledge as to where in the human body gamma globulin is produced and what mechanism is responsible for its deficiency, it might seem presumptuous to inquire into the physiologic nature of the globulin defect that exists. Several theoretical factors causing low globulin levels can be postulated, including, (1) a diminished rate of globulin formation, (2) a diminished rate of delivery of the formed globulin to the peripheral blood, (3) an increased rate of utilization in the body, (4) an increased rate of destruction or loss from the circulation, or (5) a definite combination of these factors.

Fortunately, in recent years it has been possible to perform tracer studies with I^{131} labeled gamma globulin which has enabled us to learn something along these lines. It has been demonstrated that the rate of disappearance of tagged globulin is no different in individuals with agammaglobulinemia than in normal persons. This would seemingly indicate that the basic defect is an inadequate synthesis of gamma globulin. This would, of course, bring to mind the possibility that this condition could be the result of an insufficient supply of precursor material for gamma globulin synthesis. Unfortunately, knowledge of gamma globulin precursors is incomplete, but certainly synthesis does not seem dependent upon protein intake.

In patients with certain rare types of liver disease, gamma globulin deficiency can occur even though there are high levels of protein intake. This suggests that the liver probably produces some unknown material needed for gamma globulin production. Deficiency of pyridoxine or pantothenic acid or administration of the pyridoxine antagonist, desoxypyridoxine, can result in gamma globulin deficiency in animals, but the required degree of lack of these vitamins must be severe. Administration of these substances in human patients seems entirely without effect.

CLINICAL FEATURES

Most of the important clinical features of patients with gamma globulin deficiency have already been mentioned. The salient picture which should, of course, bring about instant suspicion of this diagnosis is the presence of constantly recurring infectious episodes. The literature is full of fascinating illustrations of the various interesting clinical pictures that can result from this deficiency state. It has been mentioned that (1) many of these individuals present neutropenia and splenomegaly, (2) bronchiectasis is an extremely common finding, (3) repeated attacks of diseases which usually confer permanent immunity are frequently seen, and (4) on occasion some of these patients have had more than one episode of meningitis or septicemia.

Of more than passing interest are the cases reported by Varco and his associates[21, 22] in which skin was successfully grafted on a person with agammaglobulinemia from an entirely unrelated donor, whereas the reverse could not be accomplished. They felt that this successful homotransplant provided strong support for the concept that an immunologic mechanism was responsible for the usual failure of homotransplants in man except in monozygotic twins.

Another noteworthy case was recorded by Latimer[17] in an individual who had never shown any unusual susceptibility towards infection until after she was subjected to a breast biopsy, following which she developed a huge necrotic wound of her entire chest wall which would not heal until gamma globulin was administered, after it was discovered that she did have a globulin deficiency.

In summarizing the important clinical features that should be present to make the clinician suspect the possibility of agammaglobulinemia, the following are listed:

1. Grossly inadequate resistance to infection, as

manifested by frequently recurring and unusually severe infections.

2. Failure to develop clinical immunity after a seemingly adequate antigenic stimulus, such as a definite history of repeated attacks of a disease usually conferring permanent immunity.

3. Certain laboratory findings which confirm the diagnosis and which will be elaborated on in the section to follow.

Seltzer and his associates,[26] in discussing this problem, raise a puzzling point as to how it is possible for these patients, with no protection against infectious illnesses, to survive any time at all. Surprisingly enough, in many of these persons, including the one described in this paper, survival with this disease process extends for 35 years or more. They suggest several explanations: (1) the fact that most viral infections are self-limiting, (2) that antibodies to infectious agents are not solely in the globulin fraction, (3) that there probably is a tissue immunity not depending on circulating antibody, and (4) that extremely low levels of circulating antibody, not detectable by current laboratory methods when present, may still be sufficient to eradicate certain infectious processes. To this may be added the properdin system already discussed, as well as a report by Henschel,[19] who discovered that in one of his patients periods of agammaglobulinemia alternated with periods of hypogammaglobulinemia, during which time detectable globulin levels were present. This latter mechanism may not be an uncommon one and could be important in pointing out why these individuals may have periods of relative freedom from infection.

LABORATORY PROCEDURES FOR DIAGNOSIS

A discussion of laboratory procedures useful in establishing a diagnosis of gamma globulin deficiency seems almost superfluous, since only chemical proof is necessary to show that gamma globulin levels are depressed. However, the situation is complicated by the fact that the determination of gamma globulin levels is a complex procedure still reserved for specialized laboratories. Therefore, we are forced to use other methods which are easier and which would indirectly be useful in establishing or confirming the diagnosis. The following have been useful along these lines.

1. Methods of determining gamma globulin. The major methods of use are chemical, filter paper electrophoresis and Tiselius electrophoresis. The two former methods involve in their final steps a calorimetric reading that, at low levels of gamma globulin, is subject to considerable error. The filter paper method is subject to the greatest error of all because of the diffuse endpoint and calorimetric error. However, filter paper electrophoresis does serve its purpose well in following changes, particularly when these are performed by the same person. In recent years it has become increasingly possible through delicate immunochemical methods to demonstrate small amounts of gamma globulin in the blood; many of these patients, formerly believed to be completely lacking in gamma globulin, are now seen to have levels of 20 mg. per cent up to 300 mg. per cent. Normal gamma globulin levels are in the range of 600-1200 mg. per cent.

2. Zinc turbidity. This is a simple screening test which is usually elevated in most persons with repeated infections, yet in agammaglobulinemia no turbidity results.

3. Total proteins and A/G ratio. If agammaglobulinemia is suspected, an investigation of total and fractional proteins will be helpful. In the patient described in this report, the diagnosis was almost confirmed before electrophoretic studies were performed by the fact that the total globulin levels, as performed by the usual techniques, were 0.46 gm. per cent. It has been pointed out that in the Howe technique, which is most frequently used in determining fractional proteins, the alpha-globulins, whose levels are normal in this disease, are actually measured with the albumin fraction, whereas the beta-globulins, which are usually reduced in agammaglobulinemia, are determined along with other globulins, making for a high A/G ratio.

4. Plasma hemagglutinins. The hemagglutinins against heterologous blood group cells, which actually fall in the beta-2 globulin fraction, are low or absent in this disorder, which actually points up the fact that the deficiency is more than one of gamma globulin. Thus, individuals with type A blood do not have any appreciable titers of anti-B isoagglutinins, and those with type 0 blood do not have anti A and B isoagglutinins. This, theoretically, would make all of the persons with this disorder universal recipients.

5. Defective response to antibody challenge. One of the most useful tools in studying this disease is related to the fact that its victims are incapable of responding with antibody production to an antigenic challenge either from a naturally occurring disease or purified antigen. It has been shown that after administration of typhoid vaccine no levels of typhoid agglutinins can be obtained, after diphtheria immunization the Schick test remains positive, and no antibodies are developed in response to the injection of pneumococcal polysaccharide. Along the same lines these patients usually do not develop positive tuberculosis or histoplasmosis skin tests and have completely negative titers to practically all antigenic stimulants that can be measured.

6. Other laboratory procedures.

a. Unexpected normal results: Sometimes in these

patients one sees normal results in tests depending upon gamma globulin abnormalities. Thus, the thymol turbidity and cephalin cholesterol flocculation are normal in individuals with apparent liver disease in whom an elevation might be expected, as in the case of the patient in this discussion who had a negative cephalin cholesterol flocculation and thymol turbidity in the face of seemingly obvious hepatic dysfunction.

b. Sedimentation rate: The sedimentation rate is not influenced by gamma globulin changes since fibrinogen, the plasma protein which determines the sedimentation rate, is normal in this disease. The same is true of the C-reactive protein and other acute phase reactions which are not influenced by globulin changes.

c. Lymphocyte counts and lymph node histology: In many patients with gamma globulin deficiency lymph nodes have been observed to be rather strikingly hypoplastic, and some authors have used this as support for their belief that lymphatic tissue produces gamma globulin. Some patients with this disorder presumably have been found with lymphopenia, but this is not a consistent finding and was not so in our case.

TREATMENT OF GAMMA GLOBULIN DEFICIENCY

Naturally treatment of gamma globulin deficit consists of administration of gamma globulin. Most authors agree that if a blood level of 100-150 mg. per cent of gamma globulin is achieved, it will be sufficient to prevent further difficulty. In order to achieve such levels, 20 cc. of gamma globulin is sufficient when injected every 4 weeks in adults. In children, who have a longer half life of gamma globulin, medication in smaller amounts can be given about every 6 weeks. It is fortunate that one need not give gamma globulin in dosage sufficient to achieve normal blood levels since the normal adult would require 25 cc. of the commercial 15 per cent solution for complete replacement therapy.

The material cannot be given intravenously because of severe hypotensive reactions, whereas intramuscular doses of over 20 cc. are said to result in chills, fever, myalgia, and faintness. In the literature, dosage schedules of 0.3 cc. per pound are recommended, but this would lead to tremendous injections (30 cc. in a 100-pound individual). One report from Rome[18] points out that if gamma globulin is not available, whole blood transfusion can be given in certain patients and these will control symptoms although an added element of risk is introduced. No other therapy has been of any avail, including pyridoxine, a deficiency of which is said to lead to gamma globulin deficiency in animals, and ACTH. The use of antibiotics, both therapeutically and prophylactically, is indicated.

INCIDENCE

This disease is by no means an uncommon one, as evidenced by the steady stream of case reports published in only a few short years since the original description. Undoubtedly, each reader can bring to mind several patients he has seen in the course of his practice who were potential candidates for this diagnosis. Very few studies of actual incidence are available, but helpful along these lines is a study by Verschure[23] who did electrophoretic studies of the serums of 2300 persons and discovered 15 people (11 men and 4 women) with defective gamma globulin levels, an incidence of 0.65 per cent. The practical importance of remembering the frequency of this disorder was pointed out by Kozinn,[20] who mentioned a case of generalized vaccinia developing in an infant with globulin deficiency who had received a smallpox vaccination in New York City during the course of a mass inoculation program.

SUMMARY

A case is described of a 58-year-old white man who has a clinical picture and laboratory findings consistent with the important recently described entity which has been termed agammaglobulinemia. This process consists of an unusual susceptibility to frequently recurring infectious episodes associated with low or absent levels of gamma globulin in the blood. It is felt that this entity is of interest and significance since it definitely has many implications in the field of immunology. It is pointed out that the site of origin of gamma globulin in the body is not yet known, that there is reason to think that this disease can be legitimately divided into hereditary and acquired forms, and that the basic defect seems to be an inability of the body to synthesize new gamma globulin, although the reason for this inability is unknown. The typical clinical and laboratory findings are stressed, replacement therapy with gamma globulin is mentioned, and the not uncommon incidence of this disease is pointed out.

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Scientific progress is like mounting a ladder; each step upward is followed by a brief pause while the body regains its balance, and we can no more disregard the steps which have gone before than we could cut away the lower part of the ladder.

—O. G. Sutton

Pyloric Stenosis

Study of Surgical Treatment in 36 Cases

DONALD R. DAVIS, M.D., ***Mission***

Pyloromyotomy for congenital hypertrophic pyloric stenosis marks one of the greatest advances in abdominal surgery during the past half century.

Hirschsprung[4] is credited with the first accurate description of the disease in 1887, and immediately afterward surgeons began a search for a satisfactory method of dealing with the problem. Divulsion of the pylorus with graduated sounds introduced through a gastrostomy opening, complementary jejunostomy, pyloric resection, gastroenterostomy, and pyloroplasty of a Heinike-Mikulicz type were tried, but the high mortality rate attendant with these operations (50-75 per cent) made their use prohibitive.

In 1907 Fredet[3] introduced a technique of pyloromyotomy which included longitudinal division of hypertrophic muscle down to the mucosa, but not through it. The muscle was then sutured in a transverse direction, leaving the mucosa intact. His results were much better than those obtained with other methods used at that time.

Four years later Rammstedt,[10, 11] while attempting Fredet's operation on an emaciated infant, was unable to complete the transverse closure of the pyloric incision because of sudden collapse of the patient. He terminated the operation abruptly by returning the pylorus to the abdomen with the wound gaping and the mucosa pouting out through it, then hastily closed the abdominal wound. The patient was returned to his room in a moribund state with little hope for survival. To Rammstedt's great surprise, the patient made an uneventful and complete recovery. Entertaining the idea that simple division of the hypertrophic muscle was all that was necessary to effect a cure, Rammstedt had the courage of his convictions. He repeated the "incomplete Fredet operation" on subsequent patients and observed equally gratifying results. Thus, the Fredet operation, which embodied the basic principles of treatment, was accidentally streamlined into the classical Fredet-Rammstedt pyloromyotomy universally accepted today.

In the past it has been the belief that all patients should be given a trial of medical therapy provided, of course, the infant had not already reached a serious state. In such instance, operation was carried out as soon as the state of hydration, alkalosis, and anemia had been corrected. In recent years, particularly in American clinics, there has been a shift from this point of view in favor of early surgical treatment.

Jacoby,[5] Todd,[13] Malmberg,[8] and others have reported satisfactory results with medical treatment for this disease. In the main, medical management consists of the administration of antispasmodics (atropine, eumedrin, scopyle) before each feeding to relax the pyloric sphincter, the use of thick cereal preparations with re-feeding after vomiting, and frequent gastric lavage to remove mucus. For reasons that are obvious, an intelligent and patient mother is a necessary prerequisite for the success of such a program. Virtually all who support medical treatment look upon it as a "trial of therapy," so-to-speak, and they readily yield their patients to the surgeon when and if failure becomes evident.

Surgical treatment of congenital hypertrophic pyloric stenosis should be carried out as soon as the patient can tolerate the procedure. This report concerns patients treated by 11 surgeons in Kansas City over a 10-year period.

In view of the fact that the death rate is low in surgically treated cases, that cure is rapid and complete, and that the baby readily gains weight and strength and can soon be returned to its home, I am of the opinion these babies should be operated upon as soon as their condition permits. Early operation amounts to considerable economic saving since, after operation, hospitalization is reduced to a few days in contrast to the long and tedious programs of medical therapy which often terminate with the necessity for surgical treatment anyway.

MATERIAL

Case histories of infants operated upon for congenital hypertrophic pyloric stenosis at Bethany, Providence, and St. Margaret's Hospitals, Kansas City, during the 10-year period beginning January 1, 1946, and ending December 31, 1955, have been studied. There were 36 cases representing the work of 11 sur-

Presented before the Wyandotte County Medical Society, February 21, 1956.

Figure 1. Photograph showing typical spool-like tumorous deformity of pylorus in congenital hypertrophic pyloric stenosis. Note "avascular area" on the anterior superior aspect of pylorus.

geons. All patients were found to have a typical spool-like tumorous deformity of the pylorus (Figure 1) at operation, and all were treated with Fredet-Rammstedt pyloromyotomy (Figure 2).

Special reference was made to the patient's age, sex, clinical symptoms and signs, duration of symptoms, type of infant feeding, pre- and postoperative weight, technique of operation, complications, mortality rate, and results of surgical treatment.

RESULTS

There were 28 males and 8 females, a ratio of 3.5:1. The average age at onset of symptoms was 23.7 days, and home treatment was carried out an average of 12.7 days preliminary to admission to the hospital. The youngest patient was 14 days old, the oldest 5 months. Seven infants were breast-fed, 29 were formula-fed.

Projectile vomiting was present in 100 per cent of cases. The majority of infants had been "feeding problems" to some degree since birth, but in all cases it was possible to date the onset of their disease specifically. Dehydration was present to some degree in all cases; however, in none could it be classed as severe. Peristaltic rushes were visualized in more than half of the infants (55.5 per cent), and in 41.6 per cent an olive-sized tumor could be palpated in the right upper quadrant of the abdomen. X-ray examination with barium swallow was done in 17 cases and showed evidence of partial or complete pyloric obstruction in every instance. There were no errors in diagnosis.

Only one patient was submitted to surgery without a trial of medical therapy incorporating antispasmodics and adjustment of dietary regime. Thick cereal-containing formula preparations were used in most instances, and systemic reactions to atropine were frequent. Careful attention was paid to fluid and electrolyte balance, and patients were prepared preoperatively an average of 6.4 hospital days. Anemia was infrequently encountered, and preoperative transfusions were required in only two instances.

Inhalation anesthesia with ether, ethyl chloride, nitrous oxide, and Vinethene, alone or in combinations, was used in 66.6 per cent of cases. Inhalation anesthesia supplemented with local infiltration of the abdominal wall was used in 19.4 per cent, and in 13.9 per cent of cases local infiltration alone was used. Twenty-eight cases were approached through a right rectus muscle-splitting incision placed high over the right lobe of the liver. Five had transverse incisions, and three had subcostal incisions.

The duodenum was opened inadvertently during the course of pyloromyotomy in six cases, an incidence of 16.6 per cent. In five cases, simple closure was accomplished by sewing the mucosal rent with fine intestinal catgut. Four cases were closed longitudinally, and one case was closed in a transverse direction. In the remaining case in which the duodenum was opened, the mucosa, muscle and peritoneum were closed in layers with No. 00000 intestinal chromic catgut and silk, then the tumor was rotated 90 degrees and another pyloromyotomy was done.

One patient in whom the duodenum was opened subsequently died, but death was in no way related to this operative accident. One patient in whom simple closure was done developed a wound infection with deep dehiscence on the sixth postoperative day. The patient was returned to the operating room and the wound was secondarily repaired. The remaining four cases showed no deleterious effect whatever from accidental duodenal perforation. An omental graft was used to reinforce the site of pyloromyotomy in

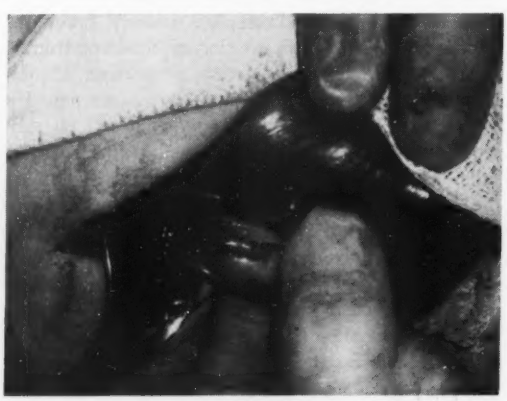

Figure 2. Photograph of completed Fredet-Rammstedt pyloromyotomy. Note mucosa pouting out through divided muscle fibers.

three patients in whom the duodenum had not been opened, and in five patients Gelfoam was used to control ooze at the site of pyloric incision.

Eighty-three per cent of patients vomited during the immediate postoperative period, but, in most instances, vomiting was minimal and of little concern. The stomachs of all babies not suffering from postoperative vomiting were aspirated at the termination of the operation.

Upper respiratory infections developed in two patients postoperatively. In one patient it culminated in post-infectious purpura hemorrhagica and death; in the other it resolved promptly and the baby recovered. Wound dehiscence occurred in five infants during the postoperative period; in two this was superficial and did not require secondary closure. One patient with deep dehiscence and two with frank eviscerations were returned to the operating room for wound repair. All patients with wound disruption completely recovered.

There were two deaths in this series, giving a mortality rate of 5.5 per cent. One patient succumbed to generalized peritonitis and multiple pulmonary abscesses. Necropsy findings indicated peritonitis was caused by laceration of the jejunum with perforation, presumably of accidental origin at the time of operation. Aspiration pneumonitis with pulmonary abscess formation also contributed to this baby's demise.

The second death was a five-month-old infant who was found at operation to have a typical spool-like tumorous deformity of the pylorus. The duodenal mucosa was opened accidentally during pyloromyotomy and closed transversely with fine chromic catgut, utilizing an omental graft to reinforce the pyloric rent. The patient developed an upper respiratory ailment during the immediate postoperative period and his condition worsened rapidly to a point where several transfusions of whole blood were required. Death ensued on the seventh postoperative day from postinfectious purpura hemorrhagica. Necropsy revealed extensive hemorrhages into the skin and subcutaneous tissues, heart, lungs, kidneys, liver, and other abdominal viscerae.

In the 34 surviving patients cure was complete. In most babies feeding formulae were tolerated within a day or two after surgery. They all gained promptly in weight and strength, and all surviving babies were in excellent condition when dismissed from the hospital.

Antibiotics were used in 83.2 per cent of cases; 29 babies received penicillin, one baby received sulfadiazine, and six babies received no antibiotic at all during the postoperative period. The average weight of babies on admission to the hospital was 7.6 pounds; on dismissal, 8.2 pounds. Patients were hospitalized an average of 17.1 days; the shortest hospital stay was four days, the longest 56 days. After pyloromyotomy, patients were dismissed after a postoperative period averaging 10.4 days.

FREDET-RAMMSTEDT PYLOROMYOTOMY

The infant usually arrives at the operating room in the arms of a pediatric nurse, its extremities having been wrapped generously in sheet-wadding and swathed liberally in blankets to conserve body heat. The baby is placed on the operating table in a supine position and secured by fixing the sheet-wadding to the operating table with strong safety pins. Conservation of body heat is of prime importance in these debilitated, dehydrated babies, and supplemental heat is provided in the form of hot water bottles, one on either side of each lower extremity and one beneath the small of the back. A number 10 or 12 F. catheter is passed through the nose to the stomach so that aspiration may be completed before anesthesia is commenced. The tube is left in place throughout the operation, and any gas that might accumulate during the operation can be readily expressed by the surgeon. By and large, ether is the anesthetic agent of choice except in institutions where a trained anesthetist is not available. In such instance, much can be said in favor of local anesthesia.

If ether is used as a part of skin preparation, care must be taken to prevent puddling beneath the baby's buttocks. The baby's tender skin can be burned with ether, and blistering can be a real nuisance during the period of recovery.

The incision preferred varies with the surgeon performing the operation. A short right rectus incision, placed high over the right lobe of the liver, is that preferred by most surgeons. The right lobe of the liver is gently retracted superiorward, and the index finger inserted into the abdomen readily verifies the diagnosis by palpation of the pyloric tumor. All intra-abdominal maneuvers are carried out with instruments which require less room than fingers.

The pylorus is delivered through the wound by gentle traction, care being taken not to drag heavily on the celiac plexus, then grasped between the thumb and forefinger of the operator. The zone of least vascularity on the anterior superior surface of the pylorus is selected, and a longitudinal incision through the serosa and superficial musculature is made. A small hemostatic forceps is used to divide the gristly muscle fibers to a point where the mucosa herniates through the pyloric wound up to the level of the serosa. The use of a binocular loupe as a visual aid at this stage of the operation will permit more accurate division of all fibers and lessen the hazard of duodenal perforation. Too, it will assist in prompt recognition of this accident, should it occur.

It is well to remember the pyloric tumor protrudes

into the duodenal lumen in a fashion analogous to the cervix protruding into the vagina. It is at this point, where the mucosa folds upon itself, that perforation will invariably occur when the operator is too energetic in splitting the duodenal end of the tumor. Robertson[12] claims it is not necessary to divide every last strand of muscle fiber in this precarious recess; he believes such a practice will not influence the incidence of postoperative recurrence. This view is not universally accepted, however. Obviously, accidental perforation must be recognized and treated, for failure to do so will result in death from peritonitis. Gentle pressure on the stomach will often force the escape of gas and stain the mucosa with bile when perforation has been committed.

Immediate closure with one or two sutures of fine arterial silk or chromic catgut is indicated. Some surgeons prefer to reinforce the rent with a tab of omentum or fat as an additional precaution. After hemostasis has been demonstrated, the pylorus is dropped back into the abdominal cavity and the liver is allowed to return to its original position. The abdomen is closed in layers with suture material of the surgeon's choice. It should be emphasized, however, that fine suture material yields more satisfactory wound healing. Ladd et al.[6] write they have found silk more satisfactory than catgut in 1,145 operated cases.

Postoperative vomiting will be minimized if the stomach is aspirated at the termination of the operation. If it appears there will be need for repeated gastric aspirations, the tube is simply left in place.

After care should include physical facilities permitting accurate temperature control and the prevention of crossed infections. Parenteral fluids are administered in amounts sufficient to supplement oral intake and maintain hydration. Eighty to 100 cc. by hypodermoclysis each day for three days is usually ample. A standard postoperative feeding program is desirable in order that interns, nurses, and ward attendants know what is expected. I do not believe it wise to permit handling of the infant without necessity until after the fourth or fifth postoperative day.

COMPLICATIONS

The most frequent complication during operation is perforation of the duodenal mucosa, and its prompt recognition and immediate repair are mandatory to preserve the life of the patient. These patients should be kept on constant gastric suction for 36 to 72 hours after operation and maintained on parenteral fluids. Ladd et al. opened the duodenum 23 times in 1,145 patients, but in all cases the accident was discovered and repaired immediately. In no case was there any untoward effect. It seems apparent that accidental perforation which is repaired promptly in the recommended fashion adds little hazard.

A disturbing situation arises when the operation is incomplete and sufficient strands of pyloric tumor remain to cause persistent stenosis of the pylorus. These patients require reoperation at the earliest possible moment with division of the remaining fibers of pyloric muscle to relieve the obstruction. In every case with persistent symptoms, incomplete operation should be corrected immediately as prompt reoperation richly increases the infant's chance for survival.

Wound infections are common, but the incidence varies widely from one clinic to another. Ladd et al. had an incidence of 2.9 per cent in 380 operated cases, whereas Nafe[9] reported 10 per cent of his 129 cases "drained during the postoperative period." Wound dehiscence, with or without evisceration, incisional hernia, and hemorrhage are less commonly encountered complications.

Gastroenteritis is by far the most dreaded complication and the most common cause of death. Looseness of stools during the postoperative period is a signal for prompt measures to bring about its control. Second only to gastroenteritis as causes of death during the postoperative period are pulmonary atelectasis, peritonitis, and bronchopneumonia.

DISCUSSION

Poor results with early pyloromyotomy in congenital hypertrophic pyloric stenosis are generally caused by inadequate knowledge of fluid and electrolyte balance. This problem having been overcome, it is now unusual for patients to have anything but a smooth postoperative course, and the family can be assured there will be no ill effects in later life. In other words, the cure is complete! Furthermore, the steady downward trend of mortality figures in recent years lends support to the assertion the operation is a relatively safe one. In skilled hands, the classical operation combined with adequate preoperative preparation yields a mortality rate of less than 3 per cent.

Levi[7] reported 100 consecutive cases of breast-fed infants who underwent pyloromyotomy without a single death; in the same period 46 bottle-fed babies were operated upon with five deaths, all from gastroenteritis. Donovan,[2] analyzing 507 cases treated with pyloromyotomy at Babies Hospital, New York, reported a mortality rate of 1.8 per cent with only two deaths in 389 cases treated during the period 1932-1946. With regard to the almost prohibitive mortality rate at the turn of the century, it has been said that in no other field of surgical endeavor has there been such a remarkable diminution in mortality statistics over a comparable period of time.

Wollstein[15] made a special study of healing after

pyloromyotomy. Using 23 necropsy specimens obtained between 24 hours and two years following operation, she determined that healing after operation is brought about by cells in the serosa and submucosa. She found the unstriped muscle of the pylorus plays no role at all in the process. Healing is complete in nine days in the pyloric wound, and the pyloric aperture is completely relaxed in two weeks. Within one month the stomach has returned to normal size, and the gap between the ends of divided muscle has practically disappeared by the end of the sixth week. When the patient reaches his second year, only a thin line of connective tissue separates the muscle ends, and the stomach is entirely normal. She concluded pyloromyotomy cures the pyloric tumor.

In 1951 Armitage and Rhind[1] reported gastric resection for perforating gastrojejunal ulcer in a 41-year-old man whose hypertrophic pyloric stenosis had been treated by gastroenterostomy at the age of 2 months. The resected specimen afforded an opportunity to examine the pylorus, and it was found that hypertrophy had persisted since infancy.

Walters[14] reported five cases in which severe gastrointestinal hemorrhage from ulcerated gastrojejunitis occurred 18 to 30 years after gastroenterostomy for congenital hypertrophic pyloric stenosis. All of his patients were treated by gastric resection, and all of the specimens revealed persistent stenosis at the point of obstruction of the pylorus.

From these reports one might conclude that in a given case of congenital hypertrophic pyloric stenosis the tumor will persist throughout life, to a degree, unless the muscle fibers are divided as in the Fredet-Rammstedt pyloromyotomy. In my opinion, the foregoing data leaves little room for medical management once the diagnosis has been established.

SUMMARY AND CONCLUSIONS

Results of surgical treatment in 36 cases of congenital hypertrophic pyloric stenosis have been presented. The material represents the experience of 11 different surgeons at Bethany, Providence, and St. Margaret's Hospitals, Kansas City, over a 10 year period, 1946-1955.

Males predominated over females by a ratio of 3.5:1, and the average age at the onset of symptoms was 23.7 days. Twenty-nine infants were formula-fed; only seven were breast-fed. Projectile vomiting was present preoperatively in all infants, and 41.6 per cent demonstrated an olive-sized tumor in the abdomen. Inhalation anesthesia was the anesthesia of choice (66.6 per cent) and a high right rectus incision was used in the majority of instances. The duodenum was inadvertently opened during the course of pyloromyotomy in six cases, an incidence of 16.6 per cent. Eighty-three per cent of patients vomited during the postoperative period, and the stomachs of all babies who did not vomit had been aspirated.

There were two deaths in this series, giving a mortality rate of 5.5 per cent. One patient succumbed to generalized peritonitis and multiple pulmonary abscesses secondary to accidental undiscovered laceration of the jejunum at operation. The second death was from post infectious purpura hemorrhagica in a patient who developed a severe upper respiratory tract infection during the immediate postoperative period. Of the 34 surviving patients, cure was rapid and complete. Patients were dismissed from the hospital after pyloromyotomy at an average of 10.4 days.

Surgical treatment of congenital hypertrophic pyloric stenosis should be carried out as soon as the patient's condition will permit. Pyloric stenosis will persist throughout life if pyloromyotomy is not performed. The operation is a safe one and cures the pyloric tumor.

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Trichobezoar

Report of a Case Involving the Stomach, Duodenum, and Jejunum in a Six-Year-Old Girl

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The following case is presented in order to show an unusual pathological specimen and interesting radiologic findings.

The patient is a six-year-old girl who was seen because of vague pains in her abdomen. For a year prior to this she had been a poor eater and had failed to maintain a normal weight for her age and height. She had had a successful operation for a ruptured appendix at the age of three. She had always been a suspicious withdrawn child, and her family had never been able to discipline or train her with good results. Up until four months before the onset of the present illness she had had a nervous habit of biting her fingernails and pulling bits of hair from her head and chewing them.

On physical examination a large, non-tender, movable mass was palpated in the epigastrium; there were no other abnormal findings. Her blood count revealed a white blood count of 16,500, red blood count of 4,100,000, hemoglobin 68%, differential count, polys 61, lymphs 34, eosinphils 1, monos 4. The sedimentation rate was 25 millimeters in an hour, Westergreen. Urinalysis showed a specific gravity 1,001; albumin, sugar, and microscopic examinations were negative. The clinical impression prior to x-ray was that of a neoplasm, probably a lymphoma, Wilm's tumor, or neuroblastoma.

The x-ray examination revealed no evidence of pathology within the barium enema study with the palpable mass lying superior to the transverse colon. The upper gastrointestinal examination immediately following the barium enema showed a convex shadow in the fundus of the stomach outlined by the magenblaze in the fundus of the stomach prior to the ingestion of barium. The ingestion of a small amount

In the case reported, physical examination and radiological studies gave evidence of a mass lying superior to the transverse colon. Surgical findings are described.

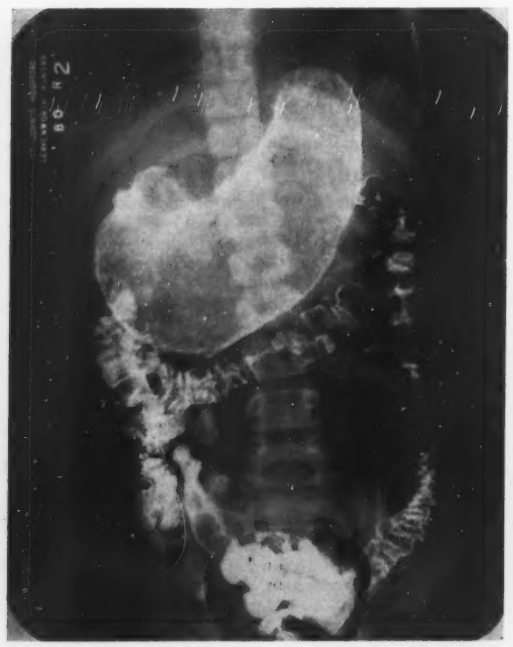

Figure 1. Film taken two hours after ingestion of meal reveals barium-impregnated bezoar in the stomach and jejunum.

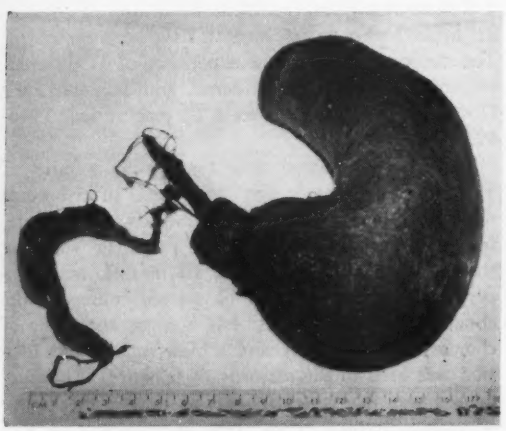

Figure 2. Photograph of the operative specimen.

of barium revealed a huge filling defect. Since the barium filtered into the surface of the filling defect, the diagnosis was trichobezoar. There was no obstruction to the ingested barium.

The irregular filling defect was also seen to extend into the duodenal cap and as far as the proximal jejunum. A two-hour follow up examination revealed the stomach empty of barium except for that impregnated in the large hair ball. It was our conclusion that the child had a large trichobezoar of the stomach, duodenum, and proximal jejunum.

On January 31, 1956, a gastrotomy was performed and a large trichobezoar forming a cast of the stomach, duodenum, and proximal jejunum was extracted. The postoperative course was uneventful.

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Rehabilitation

Restoration of Function after Fractures Is an Important and Sometimes Neglected Feature of Their Treatment

CHARLES R. ROMBOLD, M.D., *and* CLINE D. HENSLEY, JR., M.D., *Wichita*

A group of unfortunate late sequelae complicate the end results of various fractures. Some of these have been mentioned in previous sections when they were especially pertinent to the particular injury under discussion. Because of their importance, they are now brought together for consideration as complications common to more than one fracture and productive of severe and often permanent disability. These late complications which beset the path of the fracture surgeon and his patient are considered below.

LIMITED JOINT MOTION

Joint motion may be restricted by one of the following mechanisms: (1) intracapsular adhesions, (2) extracapsular soft tissue contractures, (3) adhesion of muscle to bone at a distance from the involved joint, and (4) mechanical disruption of the joint surface secondary to failure to restore anatomic alignment of the joint.

The severity and the significance of the complication depend upon the joint involved and the mechanism productive of limitation of joint motion. Where possible, prevention of this complication is the best treatment. One of the major advantages of rigid internal fixation with respect to fractures of the shaft of the femur and subtrochanteric and intertrochanteric fractures of the femur in elderly individuals is the preservation of motion in the knee and hip and the avoidance of late contractures with loss of extension at the knee. Early mobilization of adjacent joints is a dividend of skillfully performed rigid internal fixation.

Where limitation of joint motion was not or could not be prevented, restoration of function must be considered. In analyzing the approach to an individual problem, the first consideration must be the actual disability that incurs to the patient as a result of limited motion. Limited motion at the shoulder or elbow may result in comparatively little actual disability to the patient, particularly if the functional arc of motion is retained. The last 30 degrees of extension at the elbow are of little significance and do not warrant major attempts to restore full range of motion. On the other hand, loss of the last 30 degrees of extension at the knee constitutes a major disability and warrants serious effort to restore the lost range of motion.

The second factor that must be considered is the mechanism productive of the restricted motion. Intracapsular adhesions are amenable to gentle stretching, preferably carried out over a long period of time as consistent, active exercise by the patient under the

This is the concluding installment of a four-part series which began in the Journal for July. Other parts were published in the issues for August and September.

direction of the surgeon or a competent physical therapist. Rough, forceful stretching or tearing of intracapsular or extracapsular adhesions will result in hemorrhage with organization of the traumatized parts, further scar formation, and persistent contraction. Mechanical devices with a constant corrective force are frequently a major help in stretching soft tissue contractures.

Surgical release of contractures must occasionally be considered where more conservative efforts are doomed to failure. Old areas of heavy capsular fibrosis or extensive scarring of soft tissue with incarceration of the tendons about the hand are difficult problems, and it may be impossible to restore the normal gliding motion in this intricate piece of machinery. Quadriceps adhesion to the anterior femur is a fairly common complication of supracondylar fractures of the femur. The restriction of flexion of the knee results from check-rein effect of the adherent distal quadriceps and the patellar tendon. Motion may sometimes be restored across the knee by a quadricepsplasty with sacrifice of some strength of the quadriceps to regain an increased range of flexion across the knee. Conversely, extension of the knee joint may be regained by subperiosteal capsulotomy with lengthening of the hamstring tendons. Forceful wedging or manipulation of a knee flexion deformity is to be condemned since it frequently results in posterior subluxation of the tibia on the femur.

NERVE DAMAGE

Damage to the central or peripheral nervous system may result directly from the fracture in question, as an indirect complication of the treatment of the fracture, or as a late sequela of specific fractures.

Direct injury to the nervous system may complicate fractures involving the vertebral column. Neurological damage is most prone to occur in vertebral fractures above the level of the first lumbar vertebra where the spinal cord tends to fill the neural canal. Any injury which results in transient or permanent encroachment upon the neural canal may produce damage to the contents of the canal. The treatment of this major complication is complex and frequently demands the skills of the neurosurgeon, the urologist, the internist, and the physical therapist, in addition to conscientious and constant nursing care.

Immediate total paraplegia associated with fracture or fracture dislocation in the thoracic or cervical spine carries a poor prognosis. Transportation of the patient must be carried out with skill and gentleness, and the patient must be maintained in a neutral position until the mechanism of cord injury is determined and means for proper treatment are at hand. Immediate treatment is directed toward relief of spinal cord compression.

If manometric tests indicate a block, and if physical and x-ray examination corroborate this finding, it is mandatory that the compression be relieved. This may be accomplished by closed reduction of the fracture or dislocation; by hyperextension in case of thoracic vertebra, or traction with skull tongs in the case of fractures of the cervical spine; or open reduction with or without laminectomy. Open reduction is the treatment of choice with a compression fracture associated with complete forward dislocation of the superior vertebra with locking of the articular facets. Hyperextension in this injury will increase cord compression and is contraindicated.

Open reduction may also be necessary in injuries which have resulted in fragmentation of the posterior elements with displacement of bony fragments into the neural canal. Laminectomy is indicated early in those injuries in which there is evidence of a block that cannot be reduced feasibly and quickly by other means. The decision on immediate treatment is frequently difficult to make. The late psychological attitude of the paraplegic or quadriplegic patient must also be considered; if a doubt exists as to the indication of laminectomy, this consideration may tip the scales in favor of laminectomy. Where possible the counsel of an experienced neurosurgeon is to be obtained.

The late rehabilitation of the permanent paraplegic patient is another subject. This frequently is accomplished best in centers designed and equipped for that purpose. Early in the injury one must establish efficient bladder drainage and frequent turning of the patient in order to minimize urinary tract infection, urinary calculi, and the ever present menace of decubiti.

A second common direct nerve injury secondary to fractures is radial nerve damage associated with fractures of the shaft of the humerus. This complication is a result of the close proximity of the radial nerve to the middle third of the shaft of the humerus as it winds around the bone from the posterior to the anterior compartment of the arm. If spontaneous improvement does not occur, radial nerve function may sometimes be restored by neurolysis in those instances where continuity is not disrupted; or some gain may be obtained by neurorrhaphy when the nerve is divided.

Where these neurosurgical procedures fail, restoration of extensor function to the thumb and fingers can be partially achieved by transplantation of the wrist flexors to the extensor tendons. Wrist drop is best corrected by arthrodesis in the position of function.

The disability of peroneal nerve palsy may also be partially alleviated by arthrodesis of ankle joint to correct a drop foot. This provides a painless, stable joint with minimal disability and a satisfactory gait.

It eliminates the need for a brace and is compatible with a practically normal gait.

Lateral instability of the foot may be overcome with a triple arthrodesis stabilizing the subtalar joint, and this may be combined with an ankle fusion with good functional results.

Late neural complications of fractures are typified by the tardy ulnar nerve paralysis which may occur years after a fracture involving the elbow in childhood. Growth disturbances resulting in a cubitus valgus deformity will slowly produce a stretch deformity of the ulnar nerve resulting in late ulnar nerve palsy. This may be corrected by transplanting the ulnar nerve anterior to the medial epicondyle, thus shortening the route traversed by the nerve.

A painful median nerve neuritis may result from narrowing of the anterior carpal tunnel, secondary to deformity from an old Colles' fracture. There may also be loss of function of the motor branch of the median nerve with loss of opposition and abduction of the thumb. This complication can be corrected by the comparatively simple operation of division of the anterior carpal ligament. With decompression of the median nerve, function is usually rapidly restored.

ISCHEMIC CONTRACTURE

Circulatory embarrassment may complicate fractures of either the upper or lower extremity. It may be a complication of treatment rather than one resulting from the original trauma. In its most virulent form it will result in ischemic necrosis of the soft tissue and may end in severe permanent disability or amputation of the involved part. The lesion results from impairment of arterial blood supply, and in the upper extremity it may produce a typical syndrome, the end result of which is known as Volkmann's ischemic contracture.

This entity is pathologically an ischemic necrosis of muscle and nerve involving the flexor surface of the forearm with secondary contracture of the flexor muscle mass resulting in a flexion deformity of the wrist, clawing of the fingers, and variable degrees of sensory loss. As with many other complications in the treatment of fractures, prevention is the key to treatment. The complication is most prone to occur in treatment of supracondylar fractures of the humerus in children. It is this possible end result which makes it mandatory that a palpable radial pulse be present at the close of manipulation of a supracondylar fracture. If the degree of flexion at the elbow is sufficient to obliterate the radial pulse, the elbow must be extended again to the point where the pulse becomes palpable even though this may mean some loss of reduction.

If reduction cannot be maintained along with adequate circulation, traction becomes the treatment of choice, utilizing either skin traction with the elbow partially extended or skeletal traction with a Kirschner wire through the olecranon. Rehabilitation following Volkmann's ischemic contracture generally produces unsatisfactory results, with the final disability being proportional to that present at the beginning of treatment. Partial carpectomy with shortening of the forearm may be of value in releasing the contracted soft part. Wrist fusion and tendon transplants in selected cases may be of help in restoring function to the hand.

Bryant's traction in the treatment of fractures of the shaft of the femur in children is frequently not recognized as a potential source of ischemic contracture. Bryant's overhead traction is almost a universal form of treatment for fractures of the femoral shaft in patients under the age of five or six years. It is in general safe and is productive of excellent end results. It may, however, be complicated by obliteration of the popliteal artery if the knee is hyperextended in the position of traction. It is imperative that the circulatory status of the extremity distal to the knee be inspected at frequent intervals after the initial application of Bryant's traction. If the traction is in alignment in such a manner that the knee is in a position of slight flexion, there will be little chance of obliteration of the popliteal vessel.

OSTEOMYELITIS

Antibiotics have been of great help in preventing and treating infections following trauma. This fact, however, does not eliminate the threat of osteomyelitis as a complication in the treatment of compound fractures, nor does it eliminate the possibility of disaster following elective open procedures on closed fractures.

The initial treatment of a compound fracture is the most important factor in the prevention of infection. This is a matter of urgency. The first step after diagnosis and assessing the wound is to convert the wound as nearly as possible to a surgically clean wound. If this can be accomplished during the period of contamination but before the development of active infection, the wound may be considered for primary closure. In general, if a primary closure can be satisfactorily obtained, a better end result can be anticipated. It is of course not feasible to close a potentially infected compound fracture, and if it is not possible to obtain a surgically clean and debrided wound within a period of 6 to 12 hours after the injury, the wound should be left open and considered for secondary closure at a later date.

A second potential source of osteomyelitis associated with fractures is that which results from open elective procedures on closed fractures. It is a disaster to the surgeon as well as to the patient to convert a relatively simple problem of a closed fracture into an open

draining wound usually associated with nonunion and deformity of the bone with a secondary soft part deformity. This possible complication must always be weighed in the balance of the surgeon's thought when considering the feasibility of open versus closed treatment.

When confronted with established osteomyelitis complicating a fracture in an extremity, the physician must decide on treatment after consideration of the following factors: the site of infection; the extent of damage; the adequacy of skin covering; the organism and its susceptibility to antibiotics; the extent of union; the extent of systemic symptoms; the location; the duration; and the patient's wishes. Rehabilitation may be a prolonged and sometimes unrewarding process marked by numerous operations with pedicle or cross leg skin flaps, bone grafting, sequestrectomy, incision and drainage procedures, ultimately to result in amputation. The wisdom and experience of the surgeon will be taxed by such a problem, and a wise course in protecting both the surgeon and the patient will be to obtain consultation in specific instances of severe disability.

DECUBITI

A point of pressure over a bony prominence may develop either within a cast or in the course of bed rest, with secondary necrosis of the skin and soft tissues. This results in a decubitus ulcer which is usually infected and slow to heal, and the patient may focus his attention on the ulcer in spite of perfect union and function obtained in the treatment of the original injury.

A possible decubitus is always to be suspected when a patient in a cast complains of burning pain or a sensation of pressure over a bony prominence. The development of this complication is also to be feared in elderly individuals who are subjected to prolonged bed rest, particularly if they are in traction or other apparatus which prevents frequent change of position. The ulcer, once present, demands immediate relief from further pressure. Debridement is indicated in the presence of necrosis of skin and subcutaneous tissues, particularly when complicated by secondary infection. The use of any bland antibiotic ointment may be undertaken after relief of pressure and debridement of necrotic tissue. Plastic procedures may be necessary to obtain final closure of the ulcer.

PSYCHIC COMPLICATIONS OF FRACTURES

The surgeon may be forcibly reminded that he is treating an individual with a broken arm or broken leg rather than treating only a fracture. The motivation of a patient, the orientation, the basic fears and pride will frequently be determining factors in the end results. Cooperation, determination, and intelligence on the part of the patient are strong allies in the treatment of any injury. The presence of fear, false pride, or an inadequate personality may jeopardize the best efforts of the surgeon. These factors are treated and influenced by the ill defined but powerful factor of personal relationship between the surgeon and patient.

A sometimes pernicious, psychic influence in the treatment of fractures is that disease entity known as "compensationitis." Where the patient is influenced by a desire to obtain a monetary reward, or where consciously or unconsciously he lacks the driving force to return to his previous status and to make the most of the function available and to make the least of his complaints, the end result will be deleteriously affected. Again the personality and influence of the physician will be a factor in overcoming the patient's tendency to dwell on his disability and to minimize the functional capacity that remains.

FRACTURES IN CHILDREN

In the broad field of fracture rehabilitation, a separate note should be made regarding a difference in approach to the problem of fractures in adults as compared to the same injuries in children. Similar injuries in individuals of different age levels involve entirely dissimilar factors due to the presence of rapid healing in childhood and the influence of growth. Open reduction is rarely indicated in fractures of the long bones of children except for specific fractures. Fractures about the elbow joint involving the epiphysis, such as the head of the radius or the lateral condyle of the humerus, may require open reduction and anatomical reposition. The head of the radius should never be excised during its growth period. The same is true of the lateral condyle of the humerus.

Extensive physical therapy is rarely required in fractures in children. Restoration of motion is accomplished with greater ease and with normal active function on the part of the child. One may usually accept reductions that are less than anatomical in fractures in children. Rotational deviation should be corrected, and anatomical reduction of epiphyseal injuries is to be desired. Observation throughout the growth period is required when there is a possibility of deformity developing secondary to epiphyseal injury.

SUMMARY

The rehabilitation of the fracture patient is a broad field which involves many separate disciplines in the field of medicine. It may produce challenging problems and, at times, insurmountable difficulties with end results less than those desired. It offers the reward of accomplishment in the face of difficulties and provides a satisfying sense of achievement both to the patient and to the surgeon in charge.

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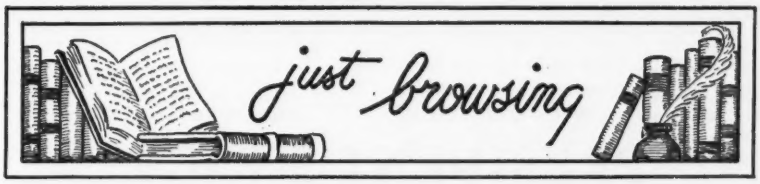

"In the last one or two decades we have got so into the habit of considering the tendency to fatal termination of a lesion, and overlooking the clinical or symptomatic and disturbing elements of the lesion, that we have forgotten the patient almost entirely. In other words, the school of pathology did not carry along with it the desire and the training for the relief of the symptoms of the patient as it progressed in its efforts to relieve him of the pathologic condition. It is hardly necessary for me to say to you that patients come to us for the relief of symptoms, for the relief of pain, discomfort, and annoyance, and a patient from whom you have removed a carcinoma of the pylorus is not so grateful to you for the result as is the man who has had an ingrowing toenail successfully removed. This is an absolute truth, and I think in that particular we have been negligent in considering the comfort of our patients both before and after operation. . . .

"This man has a dragging sensation in his scrotum. [He had a varicocele.—Ed.] He has pain in his back. He has discomfort, and when night comes he has one ache in his scrotum until the morning, and to him it is just as important to relieve that pain as it would be to relieve the pain and discomfort which are alway less in the case of sarcoma of the testicle and elsewhere, and in handling these cases we must be more considerate for the patient's ills and pains and aches. In my mind, a lack of this consideration is the reason why all the cults of medicine have originated. I believe this, that the regular family doctor is always the first choice, and it is only after his shortcomings have been grossly accentuated by his inefficiency that anybody else is called up for relief. The next decade is going to bring a constant and forceful current on the relief of the symptoms of a patient, as well as relief from the gross pathologic entity which in some cases is discernible, but in a large percentage of cases is not."

Advances in anesthesia, general availability of blood transfusions and more efficient control of infection by antibiotics have extended the field of "safe" surgery to an extent almost unimaginable even a few years ago. As a result we are participants in an era which puts the emphasis on heroic or sensational surgical procedures such as mitral commissurotomy, aortic grafting, pancreatico-duodenectomy, pneumonectomy, the separation of Siamese twins, and the like. Have we, in the course of our "progress," neglected the more common individual who has a simple, benign, curable (but uncomfortable) disease process for which he needs "relief of pain, discomfort, and annoyance"? A comparison comes to mind in wartime experiences when the "slightly wounded" soldier, having no demand on priority, was apt to be kept waiting a long time for his surgery, even to the point of developing complications which prolonged disability, although by good treatment carried out early he could usually be returned to full duty soon. Of course, we said (and correctly) that "we can't treat them all at once," and the severely injured *did* need earlier help to save life. None-the-less we should not lose sight of the fact that the relief of discomfort due to an ingrown toenail will be important to the person who has it—and he will be grateful.

But who, you ask, made the statement quoted earlier? It was not so "new" as might have been supposed, having been made by John B. Murphy* in one of his famous clinics, in 1911 —45 years ago!—*O. R. C.*

* W. B. Saunders Co., The Surgical Clinics of John B. Murphy (1912).

PRESIDENT'S PAGE

DEAR DOCTOR:

During the past month I have participated in numerous activities. I have met wonderful people and heard many interesting discussions. A day away from my office is usually rewarded by some unusual talk or person.

I have been interested in medical public relations for several years and have known for a long time that many of you are most capable performers in this field. Physicians are rather reticent about appearing before the public, but whenever they do they are usually most successful.

Recently I heard a member of our Society deliver a lecture on his trip to Russia. As I listened to his remarks I was again reminded of this wonderful country in which we live. We do not appreciate enough our freedoms such as the right to worship, school system, free enterprise, freedom of speech, and many others. What more can we do to preserve this democracy? Respect the rights that we have, know more about political issues, be active in community affairs, and exercise our right to vote when given the opportunity, thus having an integral part in this wonderful country.

Fraternally,

Clyde H. Miller M.D.

President

P.S. Be sure to exercise your voting right.

EDITORIAL COMMENT

Care for Military Dependents

The passage of Public Law 569 of the 84th Congress and its signature by President Eisenhower on June 7, 1956, may be one of the most significant health activities on the part of the federal government during the last decade. This law provides limited medical and hospital care for the dependents of servicemen. The act is known as the "Dependents' Medical Care Act."

This short act of only five pages defines a serviceman as a member of any uniformed service who is on duty for a period of more than 30 days. His dependents are wife, unremarried widow, unmarried children who are less than 21 years of age, parents or parents-in-law under certain circumstances, etc. Such dependents have previously been eligible to receive care in a service hospital. As a result of this act, which becomes effective on December 7, 1956, these dependents may receive limited medical care and hospitalization from civilian sources.

Regulations are still being formulated, but in general service facilities shall not be expanded but they shall be efficiently used. However, dependents not living with their serviceman may choose either the service hospital or civilian facilities. Dependents who live with the serviceman may generally use civilian facilities if service hospitals are crowded, and in the case of obstetrics they have their choice.

Care to which dependents are eligible is limited to include diagnosis, the treatment of acute medical and surgical conditions, the treatment of contagious diseases, immunizations, and maternity and infant care. The above are authorized generally if care is rendered in a licensed hospital. Not authorized is hospitalization in any nursing home, or any service principally requiring domiciliary care. Also not authorized are those services normally thought of as out-patient services. Excluded also are treatment for nervous and mental diseases, any chronic diseases, or a surgical procedure which is not a medical necessity. Not authorized are ambulance service, home calls, or dental care. Dependents may not receive prosthetic devices, hearing aids, orthopedic footwear, or spectacles.

Hospitalization in semi-private accommodations is authorized up to 365 days for each admission and includes all supplies and equipment, diagnostic procedures, and services that may be necessary. The patient is charged either $25 for each admission or $1.75 per day, whichever charge is the greater. That is to be paid directly to the hospital. The remainder will be paid by the government.

A tentative definition of a physician is a person who is legally qualified to prescribe and administer all drugs and to perform all surgical procedures.

According to terms of the act, the Department of Defense will negotiate with the Kansas Medical Society. The Society will submit a fee schedule which is being prepared by the Fee Schedule Committee and by the Council. The fee schedule is to be a realistic schedule of prevalent rates in Kansas. Care for dependents is not to be given for reduced payment, but neither does the government expect to pay a premium. Fees are to be realistic and are not necessarily based upon any other schedule in existence. The Society has already received a standard nomenclature consisting of 1,600 separate items for which fees shall be established.

Until this program is in operation, one can only guess the effect it will have upon the practice of medicine in this state. The present guess is that the effect will be barely noticeable. It is believed that many dependents of servicemen will continue to seek care at service hospitals and that the principal effect will be noticed in Kansas communities such as Hutchinson, Junction City, Olathe, Salina, Topeka, and Wichita, where military installations exist. Elsewhere, there will be an occasional instance of a dependent not living with a serviceman who will seek home town care. All in all, it appears the effect of this program will be slight, EXCEPT FOR THE PRECEDENT. THIS SETS THE STAGE FOR THE FUTURE.

The government has set a basis upon which it proposes to purchase medical care. It has selected to negotiate with organized medicine. It has asked, in effect, for a formula under which the doctor will provide care to whoever the government lists as its dependents.

The program had better be worked out with care because the doctors might well find themselves living under that formula for a long time to come.

The Journal Grows

Our September JOURNAL, which contained an even hundred pages, was the largest issue we've ever published. There's no merit in bigness alone, and an issue of that size was never an objective—it just happened. But we do feel that a milestone has been reached.

Most commercial publications base the number of pages for a particular issue on the amount of advertising that issue will carry. It's only good business to keep costs in line with revenue. But the JOURNAL, operating on a non-profit basis, makes no attempt to preserve an advertising-text ratio. We schedule material for publication in a certain issue

ACHROMYCIN

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[1]Posner, A. C., *et al.*; Further Observations on the Use of Tetracycline Hydrochloride in Prophylaxis and Treatment of Obstetric Infections, *Antibiotics Annual* 1954-55, pp. 594-598.

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without regard for the income to be received. It usually works out that the loss on a text-heavy issue is balanced by gain on another issue that is more lucrative. In the past ten years, the biggest issue of any year was the one carrying the most pages of advertising in only three instances.

In the years just before World War II, an issue of the JOURNAL routinely contained 52 pages. During the war, when advertisers wished to contract for more space, the size of issues was restricted by the amount of paper available, so some advertising contracts were rejected and the size of issues was usually held to the customary 52 pages. As paper supplies increased, so did the number of JOURNAL pages. A study of issue sizes during the past ten years shows the rate of JOURNAL growth.

In 1947 and 1948, the six largest issues were of 72-page size; the smallest two carried 60 pages each. The maximum in 1949 was 80 pages, and the minimum was 68. The range between extremes in 1950 was even greater, 88 pages in the biggest issue and 60 in the smallest. The high in 1951 was 84, and the low was 64.

During the past five years only eight issues have carried fewer than 72 pages, one issue each in 1951, 1953, and 1955, two in 1954, and three in 1952. In 1951 the maximum was 84 pages; in 1952, 80 pages; in 1953 and 1954, 84 pages; in 1955, 92 pages.

No seasonal pattern is evident in a study of these statistics. The biggest issue of the year did not consistently fall in any month or at any special time of year. The calendar seemed to have nothing to do either with the smallest issue of the year. December was distinctive during the past ten years in that it did not appear in either the largest issue or smallest issue column, and other months appeared in both.

Where do we go from here? We don't know, but we hope that the bigger JOURNAL has added to the interest and pleasure of its readers. And, just in case this discussion of size makes some reader curious about the number of pages in this issue, we'll give you the answer. This is printed on one of 92 pages in our October issue.

Rural Health Conference in Kansas

The Kansas Medical Society, which will celebrate in 1959 on the occasion of its 100th anniversary, will have occasion to make the year even more memorable by serving as host to a national meeting early that year. The Fourteenth National Conference on Rural Health will be held at Wichita, with headquarters at the Broadview Hotel, March 5-7. Members of the Sedgwick County Medical Society will join the state organization as hosts.

The Council on Rural Health of the American Medical Association is composed of ten physicians. Its advisory committee is made up of ten lay persons, both men and women, representing the American Farm Bureau, the Farm Foundation, the National Grange, home demonstration units, agricultural extension services, and the farm press. Mr. Aubrey D. Gates, who attended the 1956 meetings of the Kansas House of Delegates to receive the Kansas invitation for the 1959 conference, is field director of the A.M.A. council.

Through the 15 years of its existence, the council has worked to improve the health of the rural population of the nation by providing a means for exchanging ideas. The conference gives rural people an opportunity to tell what they want and expect in the way of health services, to give constructive criticism, to suggest methods of improving medical care in agricultural areas. It also provides an opportunity for physicians to discuss problems of rural health, services, and objectives, ranging from mental health to preventive medicine, infant care, geriatrics, health insurance, hospitals, etc. Although a formal program is planned for the conference, ample time is allowed for round table discussions in an informal atmosphere.

It is impossible to evaluate results of such a program, and no attempt has ever been made to list the accomplishments of this effort to improve the health of those in our farm communities. But it is apparent that a purpose is served if a better understanding of mutual problems results, if physicians and their rural patients see each other in better perspective. Kansas, as a state that is predominantly rural, is especially interested in this program.

Two Kansans, Dr. Haddon Peck of St. Francis and Dr. Conrad M. Barnes of Seneca, both of whom have served as chairman of the Kansas Medical Society Committee on Rural Health, have taken part in national programs in past years. Dr. Virgil Brown of Sabetha, present chairman of the Kansas group, will go to Purdue University this month to attend a two-day meeting sponsored by the Council on Rural Health of the A.M.A. A program of public relations for rural areas is to be discussed.

The Kansas Medical Society welcomes the opportunity of serving as host for this national meeting. Its officers and members realize that a vast amount of work will go into planning and arrangements, but they are willing to contribute time and effort to this conference which will bring approximately 1,000 persons to Kansas. And, since the meeting will be in their home state, many who have been prevented from attending such sessions in the past because of distance, will be able to be present and participate in 1959.

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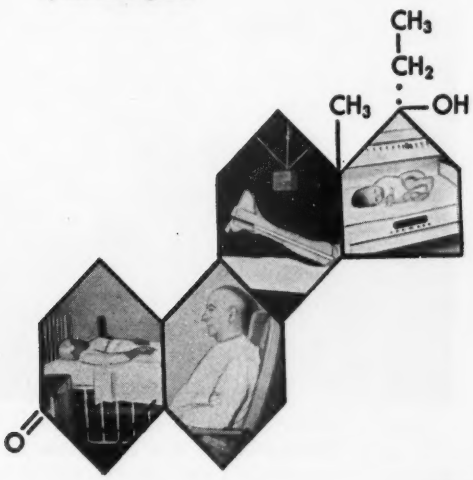

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SEARLE

Tumor Conference

Report of a Case of Meningioma in Which Complete Cure Is Probable

Edited by HOWARD P. FINK, M.D.*

Mr. Charles (junior student): This patient is a lefthanded, 42-year-old married airplane factory worker who came in the hospital on March 26, 1956, having been referred by his local doctor because of trouble reading fine print.

He had apparently been well until about three months before when the visual difficulty began. He saw his eye doctor at that time and got a pair of bifocal glasses which apparently corrected his difficulty; he felt well again for about two months, after which he again began having trouble seeing. He went back to his eye doctor, who noticed papilledema and therefore referred him here.

Besides visual difficulty he complained of occasional headaches, which he had had for approximately ten years. They were occipital and suboccipital in location, always aggravated by nervousness, and always relieved by A.S.A. compound. The only recent change in the headaches had been increased frequency. He also gave a history of a six-pound weight loss in the week previous to admission, which he attributed to anorexia caused by worry about his loss of vision.

The past history was insignificant except for slight deafness which he had had for about ten years and which was said to be otosclerotic in type. A careful physical examination was completely negative except for fresh bilateral papilledema. Routine laboratory studies all gave results within normal limits. The chest x-ray was negative.

Question from the floor: Dr. Cashion, how could you tell that the papilledema was fresh?

Dr. Cashion: The swollen nerve head was pinkish and clean looking; there were no hemorrhages and no edema residues. In older papilledema the swelling is more pronounced and the disc is dirty gray; the vessels are engorged and tortuous, and there may be organizing hemorrhages and focal edema in the retina, frequently about the macula.

Dr. Friesen (moderator): Dr. Calkins, when patients come to you and say they have difficulty in reading and want new glasses, what do you do?

Dr. Calkins: If the patient is a 42-year-old man who had noticed difficulty first with his near vision, the most probable explanation would be presbyopia, and glasses would solve his problem. However, I think any thorough examination of the eyes should always include good visualization of the fundi.

Dr. Friesen: Do you do an ophthalmoscopic examination on every patient who has trouble seeing?

Dr. Calkins: Every patient.

Dr. Friesen: The chances are, then, that this man's first doctor also did, and that the papilledema wasn't present at the first examination.

Dr. Friesen: Dr. Todd, would you show us the x-rays?

Dr. Todd: The antero-posterior projection of the

Figure 1. Antero-posterior projection of right cerebral arteriogram, showing (1) shift of the anterior cerebral arteries to the right, (2) shift of the calcified pineal body to the right, and (3) cross-filling of the left middle cerebral artery, which is deflected to the right by the tumor.

*Cancer teaching activities at the University of Kansas Medical Center are aided by grants from the National Cancer Institute, U. S. Public Health Service, and the Kansas Division of the American Cancer Society. Dr. Fink is a Trainee of the National Cancer Institute.

KNOX Protein Previews

New Study Shows Gelatine
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1. Rosenberg, S. and Oster, K. A., "Gelatine in the Treatment of Brittle Nails," *Conn. State Med. J.* 19:171-179, March 1955.
2. Tyson, T. L., *J. Invest. Dermat.* 14:323, May 1950.

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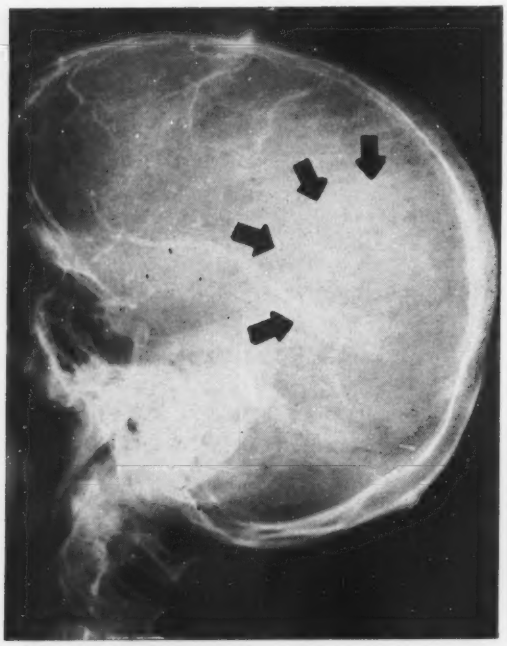

Figure 2. Lateral projection of left cerebral arteriogram, showing smudge of opaque medium (indicated by arrows) remaining in capillaries of tumor after the medium has passed through the arteries.

skull shows a shift of the calcified pineal body to the right. In the arteriograms, both the anterior cerebral arteries are also shifted slightly to the right. The right middle cerebral artery appears normal, but the left middle cerebral has an abnormal pattern; it dips medialward at one point (Figure 1). In the lateral projection a fine arteriolar plexus appears in the parieto-temporal region. In the last film, taken after the radiopaque medium had passed through the cerebral vessels, a faint smudge remains in the same area (Figure 2).

Dr. Williamson: Do you think there is increased vascularity in that area?

Dr. Todd: Yes. The smudge represents abnormal capillary filling, probably in a tumor.

Question from the floor: Is it in the right position to push the left middle cerebral artery to the right?

Dr. Williamson: Yes, but only the terminal branches of the artery.

Dr. Todd: The air studies show slight displacement of the anterior horn of the left lateral ventricle, downward and to the right.

Dr. Friesen: Apparently, then, this man's major symptoms were headaches and increasing visual difficulty, and the only significant neurological finding was fresh papilledema. The x-ray studies indicate a tumor. Dr. Williamson, would you discuss this case?

Dr. Williamson: I would like to hammer home three main points. In the first place, this man went to his doctor with the complaint of blurring of near vision, not surprising in view of his 42 years. The doctor put glasses on him and he got better. He also had the same vague, non-specific sort of headaches that many of us have, and he had had them for ten years. But, his doctor had the $50 necessary to own an ophthalmoscope and the courage to use it; and with it he discovered the patient's papilledema. If someone had not looked inside this man's eyes, the diagnosis of brain tumor would not have been made.

A second procedure available to every general practitioner is a simple x-ray of the skull. This also can establish the diagnosis of an expanding intracranial lesion by showing a shift of the pineal, provided that the pineal is calcified, as it nearly always is after the age of 25.

My third point is that a patient may have a large brain tumor and yet react normally to all neurologic tests. This is a disturbing but important fact to realize. This man's neurologic examination was completely unrevealing, and yet he had a tumor as big as a tennis ball.

These x-rays show well the refinements in brain tumor diagnosis that have been developed in the last few years. Nowadays arteriograms are of primary importance, for they will often outline a tumor accurately. The x-ray findings in this case are characteristic of a meningioma; the arteriograms show an exceedingly vascular space-occupying lesion with a smooth convex border. The mass deforms the ventricles by extrinsic pressure. These x-rays enabled us to make a preoperative diagnosis of meningioma; consequently, we could offer this man an excellent prog-

Figure 3. Arrows define the limits of tumor as it appeared at operation.

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The operation went smoothly; the tumor was easily exposed and was found to be arising from the dura over the left parietal lobe. It directly overlay the lower part of the motor cortex so that, if this man had been right-handed, he might have been aphasic. The dura bearing the tumor was excised, and the tumor emerged from the wound with only slight help from the surgeons (Figure 3). There was absolutely no surgical damage to the brain. The man was out of bed the day after surgery and went home completely well on the seventh day. He will be back at work in two weeks.

Question from the floor: How did you close the gap in the dura?

Dr. Williamson: We laid a piece of gelfilm over it.

Dr. Calkins: I think we should all remember that papilledema caused by increased intracranial pressure may very well not disturb central vision at all. A man may have papilledema of four or five diopters and enlargement of his physiologic blind spot to twice its normal size and still have 20/20 vision. When one sees an edematous nerve head, the first step in determining the cause is to check the patient's visual acuity. If this is good, the papilledema is probably due to extrinsic pressure; if it is poor, the cause is probably local disease, for example optic neuritis. Even slight inflammatory disease of the optic nerve, either at the nerve head or in the retrobulbar position, will usually affect the patient's central vision. On the other hand, choked disc unaccompanied by impairment of central vision is a finding that calls for careful further investigation, the more, perhaps, if the neurologic examination is otherwise normal, as it was in this case.

Dr. Friesen: Dr. Williamson, are meningiomas always benign?

Dr. Williamson: Not always. Malignant meningiomas are uncommon, but they do exist and may result in meningiomatosis.

Dr. Friesen: If they become sarcomatous, do they metastasize?

Dr. Stowell: Perhaps "malignant" is a better term than "sarcomatous," since the origin of these tumors is still uncertain. Meningiomas, even the benign ones, tend to grow into the overlying bone; but even malignant meningiomas rarely if ever give rise to metastases either within the cranium and spinal canal or outside. One case of metastasizing meningioma has been described by Cushing and Eisenhardt.[1] In the present case histologic examination of the tumor revealed a benign meningioma, and we can feel confident of a complete cure.

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PHYSICIANS' ACTIVITIES

Dr. George L. Thorpe, Wichita, has been named chairman of the American Medical Association Section on General Practice. He will preside at the general practice scientific assembly at the A.M.A. meeting in New York, June 3-7, 1957.

The town of Attica, which completed construction of a hospital building five months ago, announced recently that a physician had been secured for the community. **Dr. Warren J. Scherlingkamp,** a graduate of Louisiana State University School of Medicine, who had been practicing in New Orleans for seven years, opened an office in Attica last month.

Dr. Martin L. Brakebill, Sharon Springs, has resigned as physician for the Union Pacific Railroad. He will continue in private practice with an office in his home.

Dr. Bill Justus, who recently began practice in Pleasanton, has been made health officer of Linn County. **Dr. William K. Walker** is serving in the same capacity in Chautauqua County.

The Gelvin-Haughey Clinic, Concordia, announces that **Dr. Paul L. Nelson** is now a member of its staff. Dr. Nelson, a graduate of the University of Kansas School of Medicine, recently completed internship in Denver.

Dr. Herbert C. Miller, chairman of the Department of Pediatrics at the University of Kansas Medical Center, is among the founding members of the newly-organized National Council on Infant and Child Care. The council was formed to assist writers and editors in presenting developments in pediatric medicine to the public.

A feature story in the *Concordia Blade-Empire* recently paid tribute to **Dr. Guy E. Martin,** who has begun his 51st year of practice.

Dr. Perry D. Petterson, Wichita, recently be-

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came a diplomate of the American Board of Internal Medicine.

After having practiced more than 50 years in Bennington, **Dr. Llewellyn M. Hinshaw** has retired. He was honored by the community in 1952 for his long service.

Dr. Henry Laurens, Jr., formerly of Salina, joined the staff of the Scott and White Clinic, Temple, Texas, on October 1 and is specializing in gastroenterology and proctology there.

Feature stories about **Dr. C. B. Harris, Sr.,** Garnett, who has completed 50 years of practice, were carried last month in the *Kansas City Times,* the *Topeka Daily Capital,* and newspapers in the locality of Garnett. At a dinner in his honor given by the Anderson County Medical Society, Dr. Harris was given a 50-year pin. The presentation was made by his son, **Dr. C. B. Harris, Jr.**

Dr. Alfred M. Tocker and **Dr. W. G. Cauble,** Wichita, collaborated to write a paper, "Urinary Incontinence in Lerich Syndrome: Treatment by Excision of Occluded Abdominal Aorta and Common Iliac Arteries and Replacement with Homograft," published in a recent issue of *Journal of Urology.*

"Severe Radiation Accident" was the title of an exhibit presented at the September meeting of the Kansas City Southwest Clinical Society by the following physicians from Kansas City: **Dr. Peter E. Hiebert, Dr. Lewis G. Allen, Dr. William R. Allen,** and **Dr. Doris A. Kubin.**

Dr. Marshall P. Ballard, Delphos, was the subject of a feature story in a recent issue of the *Delphos Republican.*

Dr. E. N. Robertson, Concordia, recently completed 50 years in the practice of medicine and, with **Dr. Guy E. Martin** of Concordia, was honor guest at a dinner given by the staff of St. Joseph's Hospital, Concordia. Each physician was given a citation expressing appreciation for 50 years of service.

An office has been opened in Garden City by **Dr. Richard E. Hille,** who will have a general practice. He was graduated from the University of Kansas School of Medicine in 1955 and recently completed his internship at the University of Kansas Medical Center.

Dr. Chester M. Lessenden, Jr., Topeka, was one of the speakers at a meeting of the Kansas State Pediatric Society at Emporia last month. He discussed adolescent acne and tinea capitis.

Fifty years in the practice of medicine have been completed by **Dr. Walter A. Carr,** Junction City. A story about Dr. Carr was printed in the September 6 issue of the *Junction City Union.*

Dr. George L. Basham, who formerly practiced in Wichita, has opened an office in Oaklawn.

Dr. William J. Reals announces that **Dr. Peter J. Wick** is now associated with him in the practice of pathology at Wichita-St. Joseph Hospital, Wichita.

Dr. D. V. Conwell, Wichita, attended the first Pan-American Congress of Gerontology in Mexico City, September 15 to 22, and presented a paper, "The Influence of Insulin on the Renal Function of Geriatrics Patients." The Pan-American Congress is sponsored by the International Association of Gerontology and affiliated societies of the American continent.

COUNTY SOCIETIES

Dr. Robert Weber, of the Department of Medicine at the University of Kansas Medical Center, was guest speaker at the September meeting of the Shawnee County Medical Society. He discussed "Current Problems in Antibiotic Therapy." At the business session the group approved use by the Kansas State Board of Health at the Kansas Free Fair in Topeka of a device for testing blood sugar.

"The Past, Present, and Future of Blue Shield" was the subject discussed by the Wyandotte County Medical Society at a meeting held in Kansas City on September 18. Dr. Frank L. Feierabend, Kansas City, president of Kansas City (Missouri) Blue Shield, and Dr. Francis T. Collins, Topeka, president of Kansas Blue Shield, were speakers.

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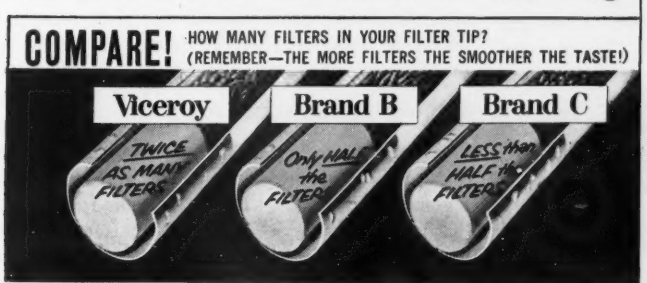
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Gastric Studies

Advantages and Limitations of Uropepsinogen Excretion Tests in Diagnosis

ROSS L. JEWELL, M.D., *Wichita*

The desirability of having a method for measuring gastric peptic activity under life stress situations in health and disease without requiring the passage of a stomach tube has stimulated much work during the past decade on a quantitative determination of the proteolytic enzyme, uropepsinogen, in timed urine specimens. This procedure is not only free of the physical and emotional discomforts of gastric suction, but is, in some conditions of psychogenic or organic illness, one of the few methods for obtaining important information.

Brücke,[9] in 1861, discovered uropepsinogen when he incubated urine with a highly acid fibrin substrate and noted digestion of the fibrin. This observation was confirmed by Grutzner[31] in 1882. Bucher,[11] in her excellent review of the development of the enzyme chemistry of uropepsinogen, states that many reports about the enzyme appeared in the German literature soon after Grutzner reported his observation. Little of the early data is fully acceptable today because of the dearth of knowledge concerning enzyme reactions and the inability of the methods available at that time to give reproducible results. This enzyme was thought to be pepsin until Glaessner,[21] in 1902, discovered that it was pepsinogen and demonstrated its conversion into pepsin. Northrop[48, 49] crystallized pepsin from swine and bovine stomachs and found this protein to act as a unit in proteolysis.

Variations of the original fibrin digestion technique for determining uropepsinogen excretion were used until 1910. A number of techniques then appeared.[11, 49] However, no significant advancements were made until Gottlieb,[24] in 1924, devised an accurate method for the simultaneous study of pepsin in the stomach, blood, and urine. He was the first worker to employ careful measurements of the pH and specific gravity of urine, to use a 24-hour urine output, and to consider the influence of diet in controlling his experiments.

In 1927 Folin and Ciocalteu[18] devised a colorimetric method for estimating tyrosine in protein hydrolysate with the use of a phenol reagent. Anson and Mirsky,[1] in 1932, devised an accurately reproducible technique for estimating peptic activity by an enzymatic splitting of carboxyhemoglobin substrate as determined by the release of a tyrosine-like substance. The test they outlined is essentially the test most widely used today. However, it has received certain modifications, e.g., Anson[2] standardized the pH at 1.6 to give the optimum enzyme reaction, and Bucher et al.[10] found that an accurate measurement of the enzyme activity in a concentrated sample could be accomplished only by dilution of the sample. Duffin and Kowlewski[15] stated that dialized urine samples should be used to obtain accurate uropepsinogen estimations. Bridgewater,[7] however, demonstrated that dialysis is unnecessary for the assay. By 1950 the unit of activity was defined[50] as a release of 0.04 mgm. of tyrosine-like substance during 30 minutes incubation at 37° centigrade.

A number of workers consider 24-hour samples necessary for accurate uropepsinogen excretion estimations.[11, 28, 57] Other workers claim similar results from specimens of shorter duration.[4, 60] Wolfson and Timms,[63] in 1954, reported that the diurnal variation of creatinine excretion matched that of uropepsinogen. On the basis that a minimum day to day variation of creatinine had previously been demonstrated by Smith,[55] they concluded that the determination of uropepsinogen:creatinine on casual urine specimens gives information similar to that from 24-hour samples.

A simple, rapid laboratory technique using the coagulation of milk as an end point and requiring no blank or colorimetric determinations was described by West et al.[60] in 1952. Although this procedure has been criticized by advocates of the hemoglobin technique, Balfour[4] stated that the test gives comparable results to those obtained by proteolytic activity and is adequate for hospital and laboratory purposes.

ORIGIN AND ROUTE OF UROPEPSINOGEN

Bucher[11] stated that the early workers postulated that pepsin was manufactured in some extragastric region and adsorbed on fibrin during its formation, or that pepsin was carried from the stomach to the kidneys by leukocytes. The stomach was demonstrated to be the source of uropepsinogen by Frouin[19] in 1904. He reported that uropepsinogen excretion had rapidly

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This is one of 11 theses, written by fourth year students at the University of Kansas School of Medicine, selected for publication by the Editorial Board from a group judged to be the best by the faculty at the school. Dr. Jewell is now serving his internship at Wesley Hospital, Wichita.

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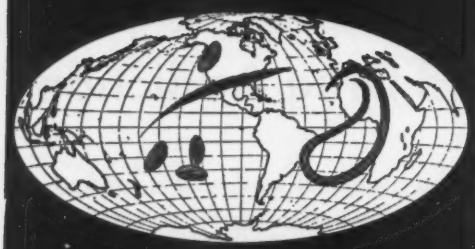

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disappeared from a totally gastrectomized dog and did not reappear when the dog was fed gastric juice from normal dogs. He also reported that a normal level of uropepsinogen excretion remained in a dog with an externalized gastric pouch in which the blood supply was kept intact. From these experiments he concluded that uropepsinogen enters the blood stream as an endocrine secretion of the peptic cells of the stomach and that no resorption occurs from the intestine. This verified the findings of Langly.[44]

Langly, in 1881, was the first to demonstrate that pepsinogen was secreted from the gastric mucosa and was rapidly converted to pepsin in the stomach. He also described the rapid breakdown of pepsin in the small intestine. Gottlieb,[24] using his improved techniques, found pepsinogen in the blood and demonstrated that its level correlated closely with gastric pepsin. Mirsky et al.,[46] in 1948, reported that the feeding of pepsin or pepsinogen, or the intravenous injection of pepsin, did not increase uropepsinogen excretion, but the intravenous injection of pepsinogen did raise the level. Herriott[34, 35] added confirmation to the evidence of a proenzyme transfer by the blood when he described the autocatalytic conversion of urinary pepsinogen to pepsin as starting at pH 5. The pepsinogen, Hirschowitz[36] stated, is carried by the blood to the kidneys where, due to its comparatively low molecular weight, it is filtered through the glomerular membrane and partially reabsorbed by the tubules.

Convincing evidence of a uropepsinogen excretion:gastric pepsin ratio of approximately 1:99 was submitted by Janowitz and Hollander.[38, 39, 40] They found no evidence that pathological changes, short of complete atrophy of the gastric mucosa, would affect the ratio and they suggested that the failure of histamine to stimulate uropepsinogen excretion is probably explained by the inability of histamine to stimulate pepsin secretion in the stomach. Similar findings have been reported by Bucher[11] and Spiro et al.[57] Gray et al.[28] however, found this correlation of gastric pepsin:uropepsinogen excretion to range from 98.0-99.5:2 0-0.5. The ratio, they stated, remained constant for each individual, and the uropepsinogen excretion was directly proportional to the amount of pepsin producing material in the stomach.[29]

RELATION TO GASTRIC ACIDITY AND TO ANTICOLINERGICS

Uropepsinogen excretion levels have been reported to be comparable to levels of gastric acid and pepsin secretion under conditions of basal secretion in normal individuals[5] as well as in persons under stress situations induced by ACTH therapy,[27] gastric and duodenal ulcers, and gastric carcinoma.[38] Podore et al.,[52] however, could find no causal relationship, and they considered the correlation to be only fortuitous. Podore's findings have been verified by many workers

who observed a loss of correlation between gastric acid and uropepsinogen excretion in a number of peptic ulcer patients,[30] in individuals treated with insulin,[29] in persons receiving caffeine or histamine,[12, 22] and occasionally in hyperchlorhydric patients.[3] Although Gray et al.[28] and Balfour[4] found no change in uropepsinogen excretion in response to anticholinergic therapy, Whitrock et al.,[61] Silver et al.,[53] and Cubberly et al.[13] noted a decrease in uropepsinogen excretion levels following methantheline and propantheline medication.

RELATION TO URINARY EXCRETION

Podore et al.[51] and Spiro et al.[57] found that the rate of excretion, volume, specific gravity, or pH of normal urine had no influence on the uropepsinogen excretion level. Balfour[4] reported no retention of uropepsinogen in patients with marked kidney disease. He pointed out that with no destruction of uropepsinogen in the urine, with no known catabolism of it in the body, and with no overflow to the kidney, any decreased level of uropepsinogen excretion demonstrates little or no production by the stomach.

UROPEPSINOGEN EXCRETION IN THE NORMAL INDIVIDUAL

Various workers have demonstrated that the levels of uropepsinogen excretion are similar to the normal gastric acid levels reported by Van Zant et al.[59] in 1932. Podore et al.[51] found children to have a lower excretion level than adults. Eastcott et al.[16] noted men to have a somewhat higher level than do women. Balfour[4] reported a lower level in older age groups. Hirschowitz,[36] however, could find no statistical difference between the younger and older adult groups.

Balfour[4] observed uropepsinogen excretion to rise slightly above normal during menstruation and pregnancy and to drop below normal levels following parturition. Most workers report no significant difference in uropepsinogen excretion levels following ordinary variations in diet, but there have been differences in the findings reported from persons subjected to marked diet changes. Balfour[4] found a lowered uropepsinogen excretion level to be associated with reduction programs and starvation. Bucher[11] and Bucher and Anderson[12] observed a marked increase of uropepsinogen excretion by doubling the protein intake for three days and a decrease caused by a similar period of low protein intake. Smith and Cowgill[54] and Goodman et al.[23] could find no variation in gastric pepsin or in uropepsinogen excretion from individuals changed from a regular diet to a high protein diet.

RESPONSE TO DISEASE

In 1955 Gray et al.[30] reported the mean value of uropepsinogen excretion to be 3670 units in normal people, 8471 units in patients with duodenal ulcer, 1815 units in patients with gastric carcinoma, and 13

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units in patients with pernicious anemia. Cubberly et al.[18] found superficial gastritis and mixed gastritis to give no conclusive variations in uropepsinogen excretion whereas a normal appearance of the gastric mucosa was not necessarily associated with a normal level.

In three of four patients in whom lymphoma involved the stomach, Balfour[4] observed an elevated uropepsinogen excretion. Spiro et al.[58] reported a surprisingly high percentage of increased blood pepsinogen levels and elevated gastric acid values in gastric ulcer patients, and he suggested that the usual clinical findings of decreased acidity may result from mucous buffers of the nonparietal secretion.

Podore et al.[51, 52] reported a definite rise of uropepsinogen excretion in the majority of duodenal ulcer patients and a much greater than normal fluctuation from the mean level; however, there was a fairly characteristic rate of uropepsinogen excretion for each ulcer patient. They stated that the gastric pepsin and uropepsinogen excretion of normal persons and those of duodenal ulcer patients have too much overlapping to have much diagnostic value when used alone but may be of value as an adjunct in the diagnosis of peptic ulcers.

Gray et al.[29] demonstrated that gastric pepsin is not significantly reduced in peptic ulcer patients following vagotomy. He pointed out that the mean average uropepsinogen excretion levels before and after vagotomy were 4923:4196 units, whereas the mean average value before and after subtotal resection was 7010:2792 units.

Hirschowitz[36] used a more complete separation of patient groups than was used by the other workers. He found that women, with or without ulcers, had normal amounts of uropepsinogen excretion, that men with duodenal ulcers of less than five years duration had normal levels, but uropepsinogen excretion was elevated in men with duodenal ulcers of more than five years duration.

Most workers have reported lower than normal levels in patients with gastric carcinoma. Gray et al.[29] observed a lower than normal mean average uropepsinogen excretion value; however, they found that 14 per cent of the levels in these patients were between 4,000 and 30,000 units, the normal being 3670 units. Within a small group of gastric carcinoma patients, Balfour[4] noted an increased postoperative survival rate in individuals who had demonstrated substantial preoperative uropepsinogen excretion levels as compared to those with low preoperative levels. He theorized that the low values were possibly caused by an inadequate protein intake as well as by the destructive efforts of carcinoma on the gastric mucosa.

All workers report lower than normal levels in achlorhydric patients, and elevated levels in the majority of patients with hypertropic gastritis.

Helmer et al.,[33] in 1932, found the peptic activity of gastric juice from patients with pernicious anemia to be about one per cent of the normal level. All workers have found blood pepsinogen and uropepsinogen excretion levels in these patients to be correspondingly low or absent in the presence or absence of stress situations.

Uropepsinogen excretion has little correlation with extragastric diseases such as gout[57] or ulcerative colitis.[6, 23, 43] Gray et al.[29] postulated that the lowered level in myxedema is secondary to the decreased adrenal function.

RELATION TO STRESS

Asher[3] stated that the most important factor in uropepsinogen excretion at any given time is the stress situation of the patient. The stress factor may have much bearing on the diurnal variation and variation from hour to hour of the levels of individuals. Asher,[3] Balfour,[4] Eastcott et al.,[16] Levine et al.,[45] and Spiro et al.[58] report variations of level during the day. Podore et al.[52] and Goodman et al.[23] reported minor to marked diurnal variation in 24-hour and overnight samples respectively. Broh-Kahn et al.,[8] however, found a fairly constant logarithmic value of uropepsinogen excretion levels throughout the day and from day to day in healthy men.

Gray et al.[28] observed a low uropepsinogen excretion level in Addison's disease and a high level associated with hyperactivity of the adrenal, pituitary, or hypothalamus in the presence or absence of the vagus nerve. They deduced from their findings that emotional and systemic stress may follow a pathway to the stomach other than the hypothalamus vagus route demonstrated by Cushing[14] and Keller.[41, 42] They theorized that "stress may cause the hypothalamus to secrete a hormonal substance which stimulates the pituitary to secrete ACTH. This activates the adrenal cortex to release a number of steroid hormones which cause the gastric glands to secrete acid and pepsinogen." They observed that acute physical stress caused a higher elevation of uropepsinogen excretion than was caused by chronic physical stress, the mean maximums being 22,000:10,000 units respectively.

A rise of uropepsinogen excretion to high levels has been observed by Balfour[4] in patients undergoing acute emotional stress. Jacobs et al.[37] found levels to decrease when psychotic patients lost contact with reality but to rise gradually when the patients recovered from insulin shock. Jacobs et al. considered that a release of adrenalin and its effect on the pituitary adrenal axis was partially responsible for the uropepsinogen excretion elevation.

During recent years there have been many reports concerning the stress effects of corticosteroids on uropepsinogen excretion levels. Gray et al.[28] pointed out that the glycocorticoid type hormones, ACTH, corti-

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sone, compound B, and compound F are effective in uropepsinogen excretion elevation. Wolfson and Timms,[64] however, noted that corticosterone, which has both glycocorticoid and mineralocorticoid activity, was ineffective in elevating the level. They concluded from this observation that the elevation cannot be closely related to glycocorticoid action. Gray et al.,[29] Spiro et al.,[57] and others[50] stated that continued ACTH administration stimulates physiologic reactions of stress in that it elicits many of the same responses as those of sustained alarm.

Gray et al.[27] observed that single large doses of ACTH or cortisone given to normal persons did not elevate the gastric acid or pepsin, but a single dose did cause marked increase in volume and acidity of gastric juice when given to a person with an inactive duodenal ulcer. A response similar to that found in the inactive duodenal ulcer patient appeared in normal persons in one to two days of repeated administrations of excess amounts of ACTH or cortisone.[25] The maximum effect of these doses in normal persons occurred in 7 to 14 days of continuous treatment.[27] This period of 7 to 14 days coincides with the usual time which precedes reactivation, perforation, or hemorrhagic manifestations of peptic ulcers during continous ACTH or cortisone therapy.[26, 32, 56]

Active duodenal ulcer patients do not display the immediate rise of uropepsinogen excretion seen in the normal person when ACTH is started. Gray et al.[25] described a delayed elevation of about five days in duodenal ulcer patients, and he postulated that this delay may denote the effect of a chronic alarm reaction on the adrenal cortex. Woldman[62] found a striking correlation between the severity of adrenal damage and the incidence of mucosal hemorrhage and ulceration of the upper gastrointestinal tract. Gray et al.[25] pointed out that the elevation of uropepsinogen excretion and blood pepsinogen was the result of peptic cell stimulation and not of renal threshold elevation. Their reasons for this hypothesis are the gradual increase for four days of the already high uropepsinogen excretion of duodenal ulcers following administration of ACTH for five days and the increase in gastric pepsin after ACTH stimulation.

Garst and Hillyard[20] observed that the adrenal pathway is more prominent in young than in older persons. These workers measured uropepsinogen excretion and 17-ketosteroids before, during, and after stressful situations and observed a marked correlation in young persons and a negative correlation in older men. They postulated, from these observations, that pathways other than the hypothalamic-pituitary-adrenal route predominate in older persons.

CONCLUSIONS

A number of limitations for the clinical use of uropepsinogen excretion approximation have been pointed out; however, the test has also been shown to have advantages.

This approximation cannot be used alone to differentiate between the normal person and the duodenal ulcer patient,[6, 23, 36] and it should not be used as an index of adrenocortico activity unless the morphologic state of the gastric mucosa is known.[13] This test is not suitable as a screening procedure for gastric carcinoma, because only 55 per cent of gastric carcinoma patients have less than 1000 units of uropepsinogen excretion.[30]

The advocates of this examination, however, suggest that it is useful within limits *as an adjunct* in the differential diagnosis of duodenal ulcer from other sources of hematemesis,[3, 16, 29] from gastric ulcer,[6, 16, 47, 52] from gastric carcinoma,[16, 29] and from other causes of dyspepsia.[16] The measurement of uropepsinogen excretion is particularly valuable in the determination of gastric peptic activity under life stress situations.[4, 6, 16, 29, 36, 45, 53] It has about the same value as measuring gastric analysis in estimating the total constant functioning activity of the stomach,[4] and normal or elevated uropepsinogen excretion appears to be of considerably more value than levels of gastric acid in the diagnosis of benign gastric lesions.[30] In certain conditions such as upper gastrointestinal hemorrhages, uropepsinogen excretion can be determined when it would be impossible to measure gastric acidity.[4]

Gastric ulcer, ulcerating carcinoma, adrenal and pituitary hypersensitivity, ACTH and cortisone therapy, and stress all possess higher than normal mean uropepsinogen excretion values, whereas gastric carcinoma, pernicious anemia, Addison's disease, myxedema, and total gastrectomy all have lower than normal mean levels.[29]

The proteolytic test is difficult to perform and requires accurate periods of collection.[6] The milk coagulation test, however, is comparatively rapid and simple to perform,[4] and it is easier to obtain urine than gastric juice and requires less of the patient's time.[4, 53]

Other criticisms of the test point toward the short term fluctuations of the uropepsinogen excretion level which require multiple observations during any test period, and toward the occasional slowly developing shifts of base line which may obscure changes resulting from experimental procedure unless multiple control periods are used.[8, 28, 53]

A uropepsinogen excretion examination at regular intervals is especially valuable in measuring gastric pepsin secretion after subtotal gastric resection,[4] and it is an aid in the diagnosis of total gastric resection,[23, 29] or pernicious anemia.[17, 23, 29]

Elevated or low values in "normal" persons may be the indication of a precurser condition to duodenal ulcer or pernicious anemia respectively.[47] It is possibly

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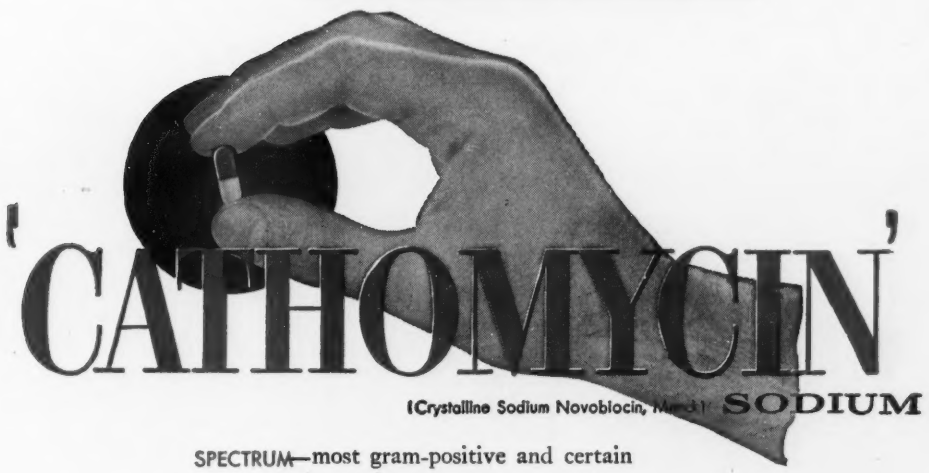

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of some value in determining the state of nutrition.[4, 11]

The examination of uropepsinogen excretion offers an opportunity to study certain drugs over periods of weeks rather than hours.[53] The determination is of much value during cortisone or ACTH therapy for the measurement of gastric response,[28, 29] or as a guide to the management of an ulcer program.[4] Some changes in functional states as related to stress and in Cushing's ulcer are comparatively apparent by the uropepsinogen excretion test. The test is also of value in judging the proper dosage of cortisone for the treatment of lymphoma.[4] It may possibly serve as a measure of studying adrenocortical situations.[16, 29, 37, 57] There is evidence that uropepsinogen excretion determination is of value in differentiating the effects of direct psychic stress from the effects of secondary stress involving adrenocortical oversecretion.[4]

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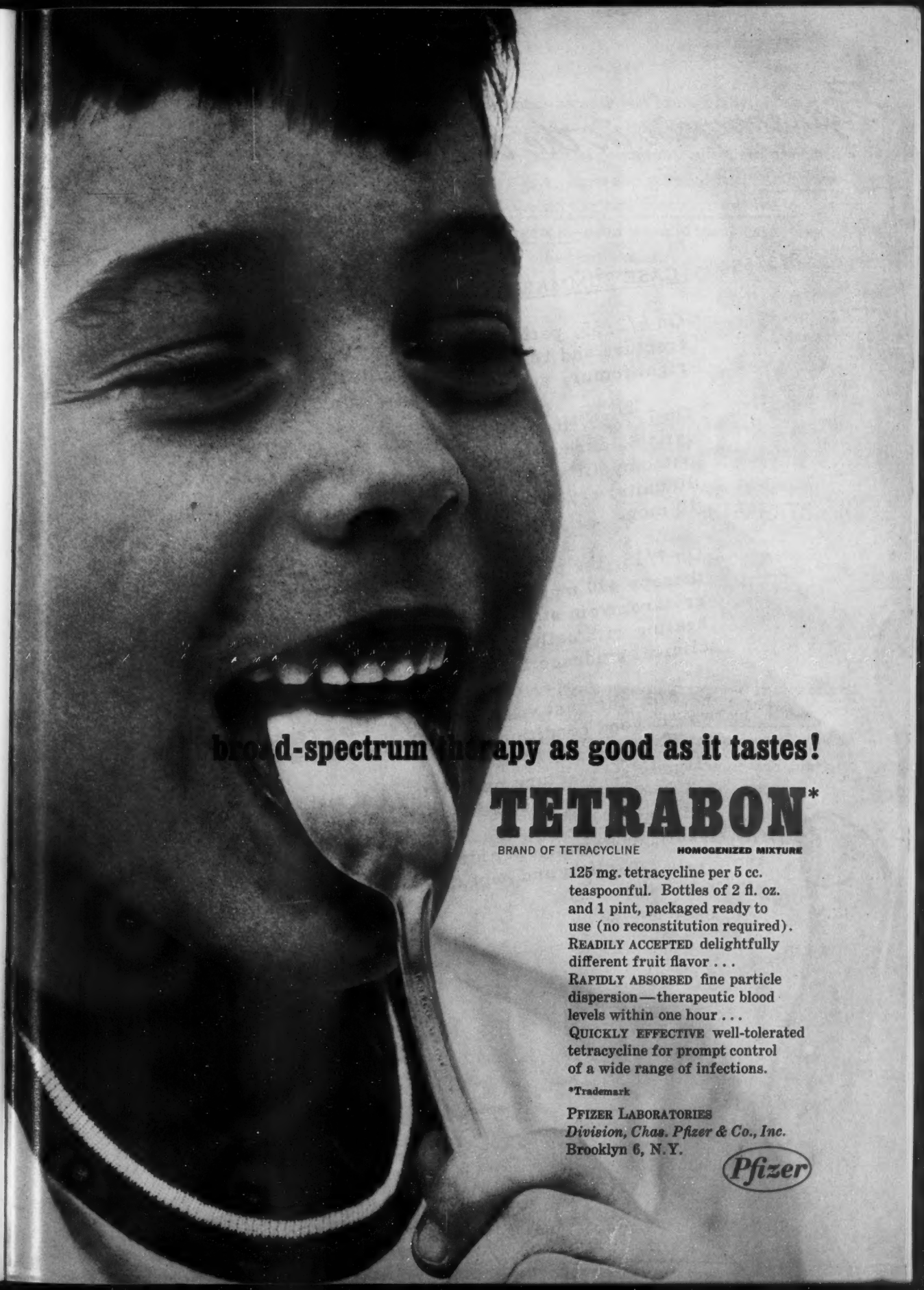
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CASE SUMMARY

On 6/2/55, patient, male, age 28, fell on an old fracture and refractured the middle third of the right femur, superimposed on an old osteomyelitis.

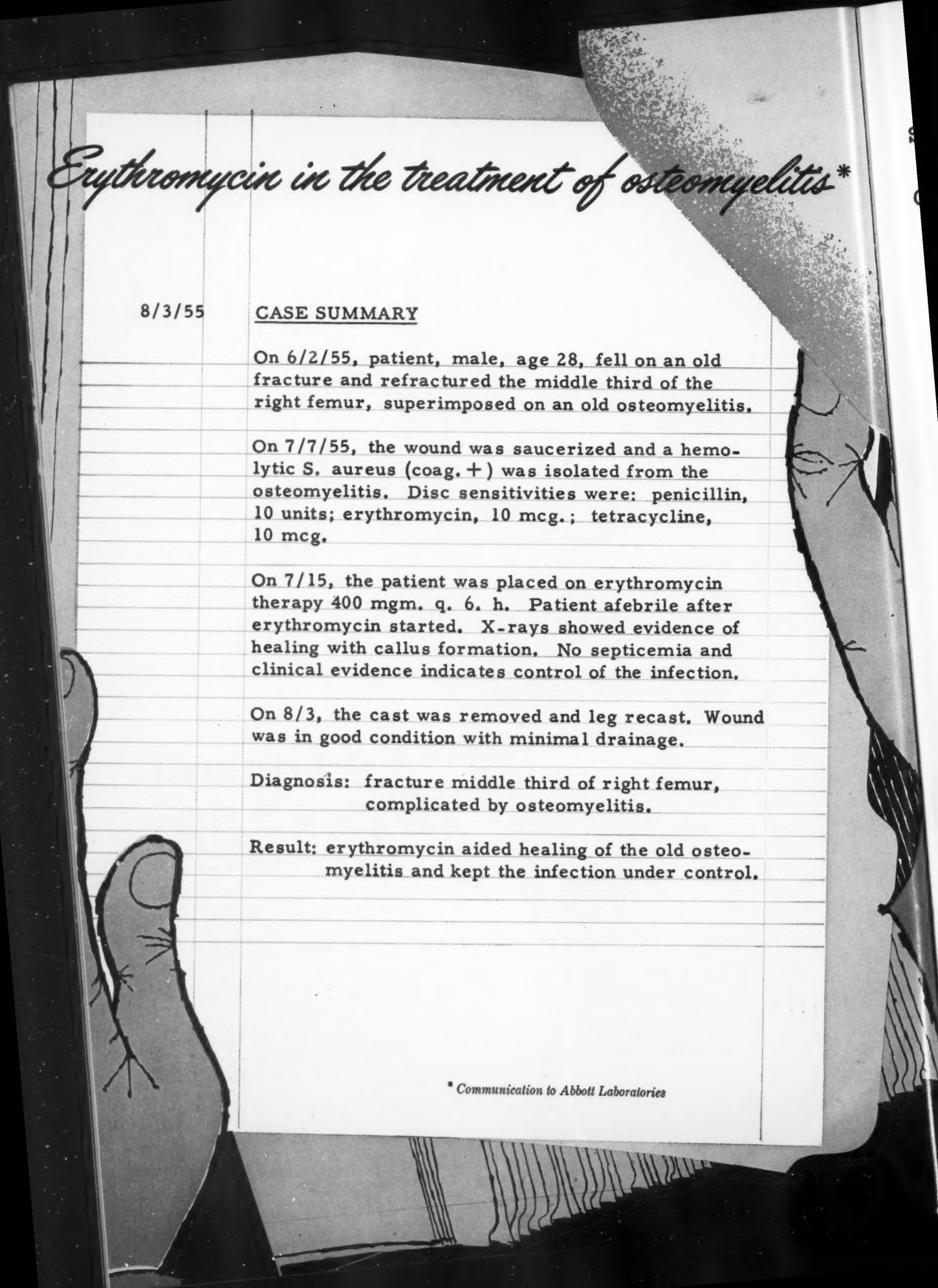
On 7/7/55, the wound was saucerized and a hemolytic S. aureus (coag. +) was isolated from the osteomyelitis. Disc sensitivities were: penicillin, 10 units; erythromycin, 10 mcg.; tetracycline, 10 mcg.

On 7/15, the patient was placed on erythromycin therapy 400 mgm. q. 6. h. Patient afebrile after erythromycin started. X-rays showed evidence of healing with callus formation. No septicemia and clinical evidence indicates control of the infection.

On 8/3, the cast was removed and leg recast. Wound was in good condition with minimal drainage.

Diagnosis: fracture middle third of right femur, complicated by osteomyelitis.

Result: erythromycin aided healing of the old osteomyelitis and kept the infection under control.



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Results of Tuberculosis Study

Recommendations of the out-of-state team of specialists who have made a study of tuberculosis in Kansas were presented by Dr. Joseph B. Stocklen, Cleveland, study chairman, at the annual meeting of the Kansas Tuberculosis and Health Association in Wichita, September 27.

"The Kansas Medical Society has followed the work of the study team with interest," said Dr. Monti L. Belot, Lawrence, president of the Kansas Trudeau Society. "Members of the Committee on Control of Tuberculosis, under the chairmanship of Dr. J. W. Spearing, as well as many individual physicians, have participated actively in the project."

Notable progress in tuberculosis control in Kansas was recognized, but Dr. Stocklen reminded doctors and health and social workers that there is much to be done if control of tuberculosis is to become a reality in the foreseeable future. The need for unified direction of public health and hospitalization aspects of the disease was stressed as essential.

Dr. Stocklen mentioned the function of private physicians in casefinding and in the treatment of tuberculosis patients. These are particularly important, he said, because of the total lack of diagnostic and treatment facilities for non-hospitalized patients in many counties and extremely limited facilities in others.

The report noted that with new treatment methods, the private physician will be called upon more and more frequently to care for patients with tuberculosis. This points up the need for continued professional familiarity with present concepts of treatment and its public health implications.

The high prevalence of tuberculosis in low income groups and the lack of public tuberculosis clinics in Kansas were revealed by the study. After consultation with the Kansas Medical Society's Committee on Control of Tuberculosis, the study team recommended that a system of medically approved clinics be established for casefinding, diagnosis, and treatment of tuberculosis.

The study report suggested the Kansas Medical Society and the Kansas State Board of Health continue collaboration in the development of a plan for the proper care and treatment of the recalcitrant tuberculous patient.

To coordinate the efforts of various agencies, the study group recommended that a tuberculosis control officer with experience in clinical and public health aspects of tuberculosis be placed in charge of the entire tuberculosis program in Kansas.

The group also recommended more frequent x-ray surveys for those in southeast Kansas and in metropolitan areas where there is the highest incidence of the disease. It was noted that the cause of control

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could be advanced by educating the public to better participation in x-ray programs.

Copies of the complete report, containing these and other recommendations, are available in limited number. Those wishing to have copies may secure them through the Kansas Medical Society, 315 West Fourth Street, Topeka, or through the Kansas Tuberculosis and Health Association, 1134 Topeka Avenue, Topeka.

THE MONTH IN WASHINGTON

Editor's Note. The following summary of Washington news was prepared by the Washington office of the A.M.A. for distribution to state and regional medical journals.

Regardless of which party organizes the next Congress or who occupies the White House, health and welfare legislation promises to take up considerable time and attention of lawmakers. There is nothing to indicate that the general subject of health has lost its appeal either to the public in general or to men who run for political office in particular.

The national platforms on which the candidates of both parties have been campaigning are somewhat of a blueprint for the type of legislation to come in the 85th Congress, convening next January 3; generally, both parties advocate more rather than less federal participation in health and welfare programs. Here are some of the points in the two platforms:

Aid to Medical Schools—The Republicans recommend "federal assistance to help built facilities to train more physicians and scientists" as a supplement to action of the 84th Congress authorizing federal grants to schools and other groups for laboratory research facilities. The Democrats state: "We pledge ourselves to initiate programs of federal financial aid, without federal controls, for medical education."

Aid to Hospital Construction—The Republican plank: "Republican leadership has enlarged federal assistance for construction of hospitals." The Democratic plank: "We pledge continuing and increased support for hospital construction programs."

Medical Research—Republicans: "We have asked the largest increase in research funds ever sought in one year to intensify attacks on cancer, mental illness, heart diseases, and other dread diseases." Democrats: "We shall continue to support vigorously all efforts, both public and private, to wage relentless war on diseases. . . . We commend the Democratic party for its leadership in obtaining greater Congressional authorizations in this field.

Vocational Rehabilitation—Republicans: "We have fully resolved to continue our steady gains in man's unending struggle against disease and disability." Democrats: "We pledge support to a vastly expanded rehabilitation program for these physically handicapped, including increased aid to states."

Medical Care—Republicans: "We have encouraged a notable expansion and improvement of voluntary health insurance, and urge that reinsurance and pooling arrangements be authorized to speed this progress." Democrats: "We pledge . . . increased federal aid to public health services, particularly in rural areas."

Social Security—Republicans: "We shall continue to seek extension and perfection of a sound social security system." Democrats: "By lowering the retirement age for women and for disabled persons, the Democratic 84th Congress pioneered two great advances in social security. . . . We shall continue our efforts to broaden and strengthen this program by increasing benefits to keep pace with improving standards of living, by raising the wage base upon which benefits depend, and by increasing benefits for each year of covered employment."

NOTES

Further evidence that federal aid to medical schools will be high on the agenda of the next Congress is the survey under way by the staff of the House Interstate and Foreign Commerce Committee. More than 50 organizations have been sent letters requesting background facts on financial needs of medical schools and the demand for medical school applicants "rather than arguments intended to support or oppose any particular form of federal aid." The information is being gathered as a preliminary to hearings in the next Congress.

Public Health Service announced the availability of 250 traineeship grants for graduate or specialized training of professional public health personnel under the newly enacted Health Amendments (Omnibus) Act. Emphasis is on bringing new and younger people into public health, men and women under 35 years of age. Congress voted $1 million for this program this year. Another 500 traineeships from a $2 million appropriation are offered for graduate nurses in administrative, supervisory, and teaching positions.

While Defense Department officials were putting the finishing touches on regulations to carry out the military dependents medical care program, the State Department was working on its own version of a program for furnishing care to about 13,500 dependents of Foreign Service personnel stationed overseas. In most instances, medical and hospital care (with a $35 deductible clause) will be supplied in U. S. military installations.

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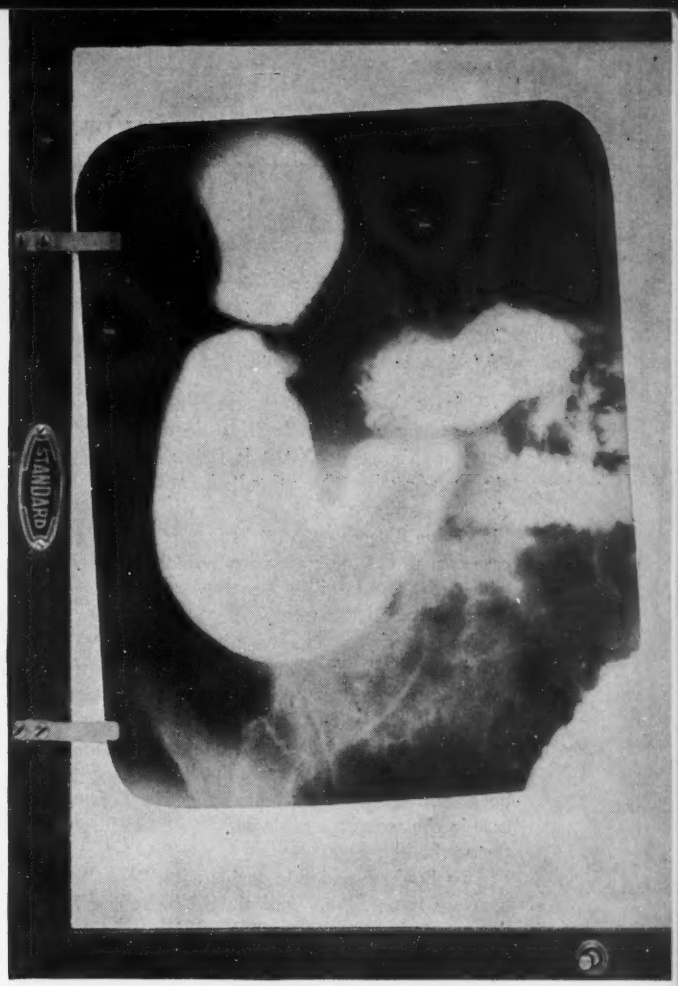

"...functional nervousness, including fatigue and anxiety, was by far the greatest detectable cause of recurrences of peptic ulcer symptoms, and in many instances it seemed likely that the same etiological factors were initially responsible for the ulcer."[1]

Peptic ulcer is a combination of the emotional and the physical. For total management, a combination of measures is often indicated. EQUANIL adds to the adequacy of routine treatment by countering psychic stress as a stimulant to vagal activity. It combats the anxiety and tension, and encourages restful sleep.[2]

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1. Weiss, E., and English, O.S.: Psychosomatic Medicine. W. B. Saunders Co., Philadelphia, 1949, p. 358.
2. Lemere, F.: Northwest Med. *54*:1098 (Oct.) 1955.

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BOOK REVIEWS

The Recovery Room—Immediate Postoperative Management. By Max S. Sadove, M.D., and James H. Cross, M.D. Published by W. B. Saunders Company, Philadelphia. 597 pages. Price $12.

This book opens up an entirely new concept of management of critically ill patients. Though it is stated on the cover that it is written about immediate postoperative management, it proceeds in its first chapter to discuss and describe what is termed an "Intensive Therapy Unit." This unit is designed to care for patients emerging from anesthesia and those who are critically ill—comatose, cardiac, neurosurgical, and accident cases.

The book is an exhaustive and detailed collection of ideas on management of critically ill patients of every category. It is well written and informative. If there could be any criticism, it might be that the authors left nursing care in the recovery room to the last and gave it comparatively little space. It has been our observation that the quality of nursing care in the recovery unit is one of the most important factors in its success.

This book should be considered a "must" in the recovery room library and should be referred to by doctors and nurses alike in operating existing units and in planning and expanding new facilities.—*W.O.M.*

Diseases of the Chest. By Corwin Hinshaw, M.D., and L. Henry Garland, M.D. Published by W. B. Saunders Company, Philadelphia. 727 pages. 634 illustrations. Price $15.

The authors have presented a well organized survey of the diseases of the chest exclusive of heart disease. They have most successfully made this a contemporary book with an inclusion of the latest developments in the field evaluated in the light of their extensive experience.

The initial section of the book involving diagnostic procedures may occasionally seemingly refute some of our classic conceptions; for example, the authors advised that the physician have the x-ray image in mind *prior* to the physical examination of the chest. As one reads on, however, there remains no doubt about the thoroughness of the authors' technics of physical diagnosis.

The apparent bulk of the volume is diluted by extremely frequent and well chosen x-ray pictures. The print is easily read. The outlines preceding each chapter and the summaries following are quite helpful.

I feel this book is a useful reference volume, and its main worth will be in the readiness with which current views in the field of chest disease may be found in an office or hospital library.—*J.L.M.*

A Modern Pilgrim's Progress for Diabetics. By Garfield G. Duncan, M.D. Published by W. B. Saunders Company, Philadelphia. 222 pages. Price $2.50.

This compact book is small enough to fit conveniently in a suit coat pocket. It is composed of two parts. The first part (144 pages) is about the experiences of a young diabetic social worker who is employed in a diabetic clinic. The remainder of the 222-page book is devoted primarily to an appendix with a short glossary and index.

Problems faced by all young diabetics are covered in an interesting fashion in the social worker's own life. In the clinic she meets all the typical types of diabetic patients. These are all treated as personalities and are given names so that their characteristics may be better remembered and understood by a new or old diabetic who reads the book. During the course of the story, explanations are made regarding blood sugar, heredity, insulin, and other subjects. Footnotes indicate where to find more details on a given problem in the appendix. Cases of insulin reactions, diabetic acidosis, gangrene, neuritis, intermittent claudication, and other complications are illustrated. Finally the patient marries and has a baby, giving an opportunity to cover the problem of diabetes and pregnancy.

The appendix gives detailed descriptions of what diabetics should know about diabetes. The various insulins and their administration and the care of insulin reactions are discussed. Testing of urine for sugar and ketones and diabetic coma receive detailed attention. Diets are explained, and the exchange system is thoroughly covered. In the glossary, definitions of such terms as calorie, fat atrophy, and Sucaryl are given.

In the index one can find quick reference to where a subject is covered both in the story and in the appendix.

This book deserves a thorough clinical trial both as a ready reference book for the physician and as a book of instruction for the diabetic patient.—*T.J.L.*

The Neuroses in Clinical Practice. By Henry P. Laughlin, M.D. Published by W. B. Saunders Company, Philadelphia. 802 pages. Price $12.50.

This volume sets forth the neurotic reactions in a readable, lucid, well-constructed arrangement. The author's modest hopes expressed in the preface are

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1. Cronheim, G., and Toekes, I. M.: Comparison of Sedative Properties of Single Alkaloids of Rauwolfia and Their Mixtures, Meet. Am. Soc. Pharmacol. & Exper. Therap., Iowa City, Iowa, Sept. 5, 1955.

2. Moyer, J. H.; Dennis, E., and Ford, R.: Drug Therapy (Rauwolfia) of Hypertension. II. A Comparative Study of Different Extracts of Rauwolfia When Each Is Used Alone (Orally) for Therapy of Ambulatory Patients with Hypertension, A.M.A. Arch. Int. Med. *96*:530 (Oct.) 1955.

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exceeded because he has successfully combined several essential elements: extensive and interesting discussions of the subject matter, sufficient historical references to orient his presentation, and ample bibliography to indicate the amount of background effort entering into such a work.

We learned medicine in the concise, comfortable etiology-diagnosis-therapy-prognosis pattern and have always had trouble fitting the psychiatric entities into the same nice pattern. We were heartened, therefore, to find that the author has given us a practical, usable reference with due allowance for the infinite variety of mental reactions.

He performs one service if no other: the inclusion of a glossary of psychiatric terms. We have always had a suspicion that we "somatically-oriented" characters were not so far removed from our psychiatric brethren if we only knew what all those words really meant.—*D.E.G.*

The Doctor's Marital Guide for Patients. By Bernard R. Greenblat, M.D. Published by the Budlong Press, Chicago. 88 pages. Sample copies free to physicians. Patient price $1.50.

This is a condensed presentation on the alleged facts of life and seems to us to be well written and as adequate for the orientation of young couples as the more involved books. After all, there is a limit to how much one can learn out of books. There is also a limit to the information a physician can get over to a nervous young couple in a reasonable period of time, and a manual of this sort for later perusal makes an excellent adjunct to his remarks. There is also a Catholic edition with obvious differences in reference to the matter of contraception. Perhaps the best recommendation is that both of the reviewer's copies were lent to prospective brides and have never been returned.—*D.E.G.*

Sleep. By Marie Carmichael Stopes, D.Sc., Ph.D. Published by the Philosophical Library, Inc., New York. 154 pages. Price $3.00.

This reviewer first felt sympathy for the author, an English woman so sensitive that she once recognized a variation of four degrees from true north in the placement of her bed. Next came a feeling of irritation for one who can find so much about which to complain in our present civilization. Finally came a feeling of amusement and the thought that some of the cures proposed are less attractive than the complaint.

It seems incredible that the author can seriously mean the following statements: "The soft foam rubber mattress is an example of a modern 'advance' to be avoided by all who value their health. It is pernicious . . . because rubber is an insulator, and cuts you off from electric currents of the earth with which you should be in contact." "It [leg cramp] was brought . . . by the stink given off by my new rubber hot-water bottle." "Till he is 15 or 16, no child should do brain work after that time [four o'clock in the afternoon]. "*No* child should be waked, not even a child of school age. Sleep is of far more value to a child than lessons." "If, on the other hand, as is so often and so foolishly the plan in modern homes, the sleeper has no chamber pot handy under or beside his bed . . . he will be very thoroughly awakened. I have seen no protest by any doctor, nor any public statement anywhere about the evil effects of this retrograde innovation in banishing the bedroom chamber, now so rampant among us masquerading as 'modern hygiene.' "

The author's pleasure in derogatory statements about this country is seen in the following: "About half the people in the United States of America suffer from sleeplessness. One is tempted to say, 'It serves them jolly well right.' They infringe the most fundamental laws of nature more than any other nation, and the ever-increasing tempo of their lives, combined with a mounting fear of future conditions, creates exactly the type of mind to defeat sleep's gentle ministrations."

It also creates the type of mind to predict that this book will enjoy little popularity in the United States. One is tempted to say of its author, "And it serves her jolly well right."—*P.F.*

Electrocardiography—Fundamentals and Clinical Application. Second Edition. By Louis Wolff, M.D. Published by W. B. Saunders Company, Philadelphia. 522 pages, 109 figures. Price $11.50.

The first edition of this text was among the earliest to substitute a rational basis of electrocardiographic interpretation for the pattern memorization method. The second edition preserves the simplicity and understandability of the original introduction which included just enough electrophysics to permit a logical explanation of the various deflections of the normal and abnormal electrocardiogram.

The author has brought the text up to date by skillfully weaving in the concepts of vectorcardiography and ventricular gradients and pointing out the additional information they may yield. His presentation of vectorcardiography is purposely superficial on the basis that its applicability to clinical electrocardiography has not yet been clearly defined.

In addition to the chapters on right and left ventricular hypertrophy, right and left bundle branch block, myocardial infarction, and coronary artery

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disease included in the first edition, there have been added chapters on arrhythmias, pericarditis, pulmonary embolism, and abnormal electrolyte patterns.

The simplicity of language and the conciseness of this book make it an ideal introduction to electrocardiography for the novice, yet there are helpful facts which make it worth while reading for the more advanced student as well.—*R.T.C.*

Christopher's Textbook of Surgery. Sixth Edition. Edited by Loyal Davis, M.D. Published by W. B. Saunders Company, Philadelphia. 1484 pages, 1359 illustrations on 716 figures. Price $15.50.

For 20 years *Christopher's Textbook of Surgery* has been a popular and standard single-volume text. The present volume is a worthy successor and is the first to appear under the editorship of Loyal Davis.

Recognizing the futility of attempting to put everything in one volume, ". . . Each contributor to this edition has had the aim to present his subject in such a way that he will stimulate the young and older students of surgery alike to read further, to think through the problems presented by each of their patients, and to ascertain by themselves the anatomic, physiologic, bacteriologic, pathologic, biochemic and pharmacologic factors involved. Surgery is the *art* of the application of the basic medical sciences and each individual engaged in its complexities of judgment, responsibility and decision must continue to educate and improve himself without thought of an end point.

"Each contributor has written in his own style of teaching and each one is authoritative in his field; each hopes that he will create in the reader a desire to learn more about the subject presented. Reading references have been provided to encourage the inquisitive and imaginative to seek further."

In keeping with the changing scope of surgical practice, some chapters, such as those on thoracic surgery and vascular surgery, have been expanded, but not to the exclusion of proper emphasis on common conditions such as hernia, gastrointestinal tract, biliary tract, and fractures. An introductory chapter on History of Surgery and a final one on Qualifications of a Surgeon round out a well balanced volume. The book deserves and will continue to be a most popular text for medical students and practicing surgeons.—*O.R.C.*

Great ideals and principles do not live from generation to generation just because they are right, nor even because they have been carefully legislated. Ideals and principles continue from generation to generation only when they are built into the hearts of the children as they grow up.—*George S. Benson.*

ANNOUNCEMENTS

Twenty-sixth annual clinical conference, Oklahoma City Clinical Society, Oklahoma City, October 22-25. Write 503 Medical Arts Building for program. Credit under category 1 by American Academy of General Practice.

Five short-term courses related to heart and circulatory system offered by New York University Postgraduate Medical School during November. Covered are cardiac roentgenology, electrocardiography, clinical electrocardiographic interpretation, peripheral vascular diseases, cardiac arrest and resuscitation. Write the Dean, 550 Forst Avenue, New York 16, New York.

December 1, deadline for submitting applications to National Foundation for Infantile Paralysis for postdoctoral fellowships. Fellowships available in rehabilitation, psychiatry, orthopedics, management of poliomyelitis, preventive medicine, research, and academic medicine. Stipends from $3,600 to $6,000. Write Division of Professional Education, National Foundation, 120 Broadway, New York City.

Course in practical electrocardiology presented by Dr. Demetria Sodi-Pallares, Mexico City, under auspices of University of Texas Postgraduate School of Medicine and Baylor University College of Medicine, Houston, December 3-7. Write Postgraduate School of Medicine, Texas Medical Center, Houston 25, Texas.

Award of $500 for best article by a physician on any subject of medical economics, $100 to $300 for other articles accepted. Deadline December 31, 1956. Address Awards Editor, Medical Economics, Oradell, New Jersey.

Next scheduled examination (Part I), written, and review of case histories for candidates of American Board of Obstetrics and Gynecology, Inc., in various cities, February 1, 1957. Case reports due within 30 days of notification of eligibility to Part I. Deadline for requests for re-examination in Part II, February 1. Write the Secretary, Robert L. Faulkner, M.D., 2105 Adelbert Road, Cleveland 6, Ohio.

Eleventh annual symposium on fundamental cancer research, University of Texas M. D. Anderson Hospital and Tumor Institute, Houston, March 7-9, 1957. Subject, "Viruses and Tumor Growth."

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A.M.A. Clinical Session

The A.M.A. clinical session will be held in Seattle, November 27-30, with headquarters at the Olympic Hotel where House of Delegates and committee meetings will be held.

The scientific program at the Seattle Civic Auditorium will consist of 45 papers beamed at the general practitioner and covering such subjects as fluid balance, urological problems, office psychiatry, varicose veins, fractures, diabetes, and heart disease. Television clinics, both operative and non-operative, will treat of anesthesia, burns, bleeding problems, intestinal obstruction, caesarean section, hand surgery, and vein stripping.

The problem of darkness does not exist for a man gazing at the stars. No doubt the darkness is there, fundamental, pervasive and unconquerable except at the pinpoints where the stars twinkle; but the problem is not why there is such darkness, but what is the light that breaks through it so remarkably; granting this light, why we have eyes to see it and hearts to be gladdened by it.—*George Santayana.*

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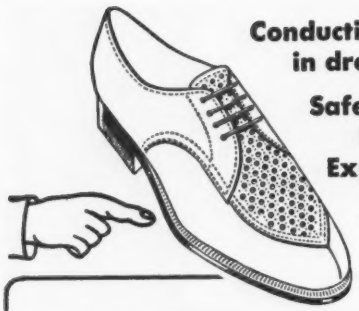

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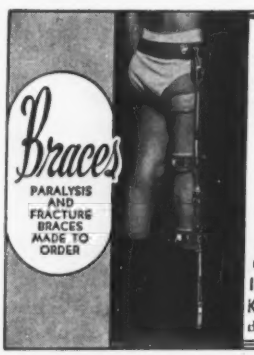

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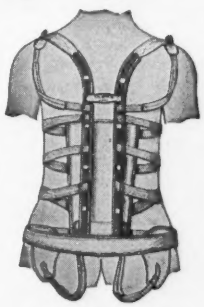

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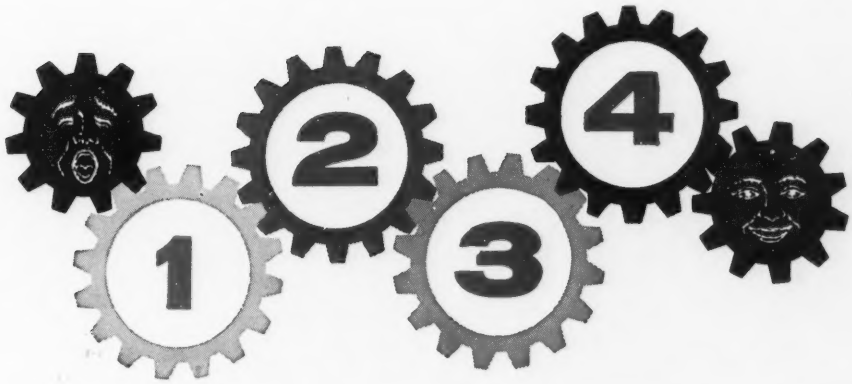

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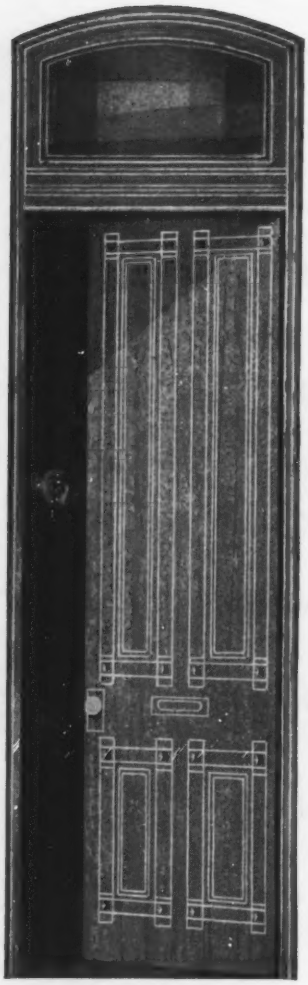

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